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Essentials of

Pathological Anatomy

Dr. O. Bollinger

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ATLAS AND ESSENTIALS

OF

PATHOLOGICAL ANATOMY

BY

DR. O. BOLLINGER

OBERMEDICINALRAT AND PROFESSOR

VOLUME II.

URINARY APPARATUS, SEXUAL ORGANS, NERVOUS SYSTEM,
AND BONES

WITH 63 COLORED FIGURES UPON 52 PLATES AND 17
ILLUSTRATIONS IN THE TEXT

NEW YORK

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1898

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LIST OF PLATES.

ILLUSTRATIONS IN THE TEXT.

Tab. 1.

Explanation of Plate 1.

CONTRACTED KIDNEY ASSOCIATED WITH A CONDITION
OF CHRONIC CONGESTION. CYANOTIC INDURA-
TION, WITH SECONDARY, UNEQUALLY DISTRIBUTED,
GRANULAR ATROPHY.

The kidney, which originally was somewhat enlarged, is now (as here pictured) reduced in size, as a result of secondary contraction, to about the normal proportions. (Weight of both kidneys = 300 gm.) The fibrous capsule was removed with comparative ease. The surface underneath it was found to be somewhat uneven, partly on account of the presence of depressions of varying depths (the effect of retraction of the tissues), and partly on account of small-sized granulations. Consistence of the organ greatly increased. Color of a dark livid hue. When cut open the kidney presented, upon the surface of the section, the following appearance : Cortex somewhat diminished in breadth, of a cyanotic hue, and separated from the region of the pyramids—which is even more deeply cyanotic—by a sharp boundary line. Abundant development of fat at the hilus.

From the history of the case it appeared that the patient, a sculptor, sixty-two years of age, had been under surgical treatment for a small ulcer upon his foot. Up to three days before his death he had been going about, apparently in good health. Then he was suddenly taken ill with difficulty in breathing and a sensation of pressure in the region of the stomach. On physical examination it was found that the heart was enlarged, the valvular sounds were clear but feeble, and the pulse was irregular, small, and easily compressible.

The urine was scanty and contained only a little albumin. There was no dropsy nor any fever. There was no appetite. His face was very pale and his finger nails were cyanotic. Death followed speedily, under manifestations of heart weakness.

The apparent cause of death was found to be idiopathic hypertrophy and dilatation of the heart, of alcoholic origin. The organ weighed 470 gm. The other pathological conditions found were the following : Congestive enlargement of the spleen ; nutmeg liver; chronic congestive catarrh of the stomach ; gall stones ; at the apices of the lungs, on both sides, evidence of former tuberculous disease. (No. 3, 1895.)

Explanation of Plate 2.

The substance of the kidney is almost entirely con-
verted into opaque yellowish masses, which in some
places (in the lower part of the picture, for example)
completely replace the cortical and the medullary sub-
stances, while in other places they occur in the form of
isolated wedges. The margins of the organ show a
slightly reddish hue. Embedded in the fat at the hilus
may be seen transverse and oblique sections of the
pathogenic emboli which plug the arterial branches.
Elsewhere throughout the kidney—although these lesions
do not appear in the colored plate—may be seen a num-
ber of spots where cicatricial contractions (the remains
of earlier embolic processes which have run their course,
have healed, and have then undergone absorption) have
taken place. At the post-mortem examination marasmic
thrombi were found in the left auricle of the heart, and
thrombi were also found attached to the inner wall of
the thoracic aorta, particularly in the vicinity of the
arch; both of which lesions are to be considered as the
products of embolic plugs that have found their way into
the general circulation after passing through that of the
kidney. The inner coat of the aorta was found to pre-
sent alterations to which the term calcareous and ulcerous
endarteritis is usually applied.

Among the other pathological changes observed in this
patient (aged fifty-nine) were the following: Marked
hypertrophy and dilatation of the heart, which weighed
490 gm., or 190 gm. in excess of the normal weight
(death, it should be stated, was due to heart failure);
anæmic infarction of the spleen; terminal sero-fibrinous
pleuritis and hydrothorax (hydrothorax inflammatorius)
involving both sides of the chest; and a moderate degree
of general dropsy. (No. 137, 1895.)

Tab. 2.
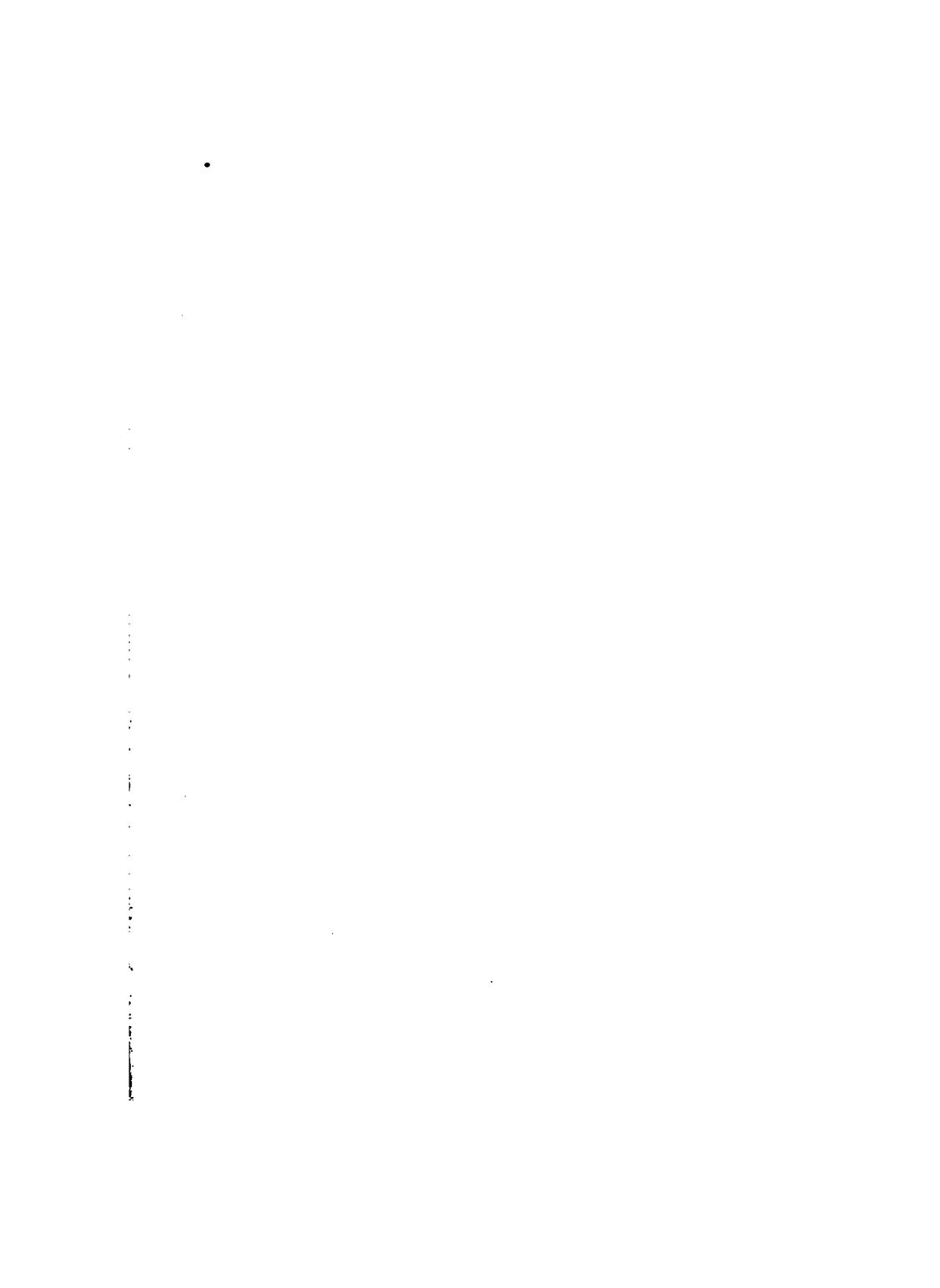
Tab. 3.

Explanation of Plate 3.

MULTIPLE SUPPURATING EMBOLIC INFARCTIONS OF THE
KIDNEYS, IN SEPTICO-PYÆMIA.

The removal of the capsule brought to view three
small foci and one large area, which, when incised,
proved to be collections of thick creamy pus. The
smaller foci, which varied in size from that of a pin's
head to that of a hempseed, were of a grayish-yellow
color surrounded by a zone of red. The larger area,
which was of a yellowish-white color, was also sur-
rounded by a broad zone of tissue of a hemorrhagic,
deep-red hue. All of these collections were embedded
in the cortical substance.

Explanation of Plate 4.

CHRONIC INTERSTITIAL NEPHRITIS: INFLAMMATORY, IN PART SLIGHTLY GRANULAR AND IN PART SMOOTH, CONTRACTED KIDNEY.

The right kidney is distinctly diminished in size and its capsule is hard to remove. In fact, small bits of cortex tissue are commonly found adhering to the inner surface of the fibrous capsule. The organ presents a pale coloring and in some places it is a little spotted. The surface at some points is finely granular, while at others it is smooth but depressed, as if drawn in. The substance of the kidney is tough and cuts like India rubber. The line of demarcation between the cortex and the medullary substance is plainly marked. The cortical zone is reduced in breadth, but not to the same degree at all points; it is noticeably paler than are the darker and somewhat shortened pyramids. Weight = 120 gm. The left kidney, which weighed only 65 gm., showed alterations similar to those described above but of a more pronounced character.

In this patient (a hostler, twenty-seven years of age) various other pathological changes were found, as, for example: Marked hypertrophy and dilatation of the heart (weight = 620 gm.), particularly of the left ventricle; a terminal, acute, sero-fibrinous pericarditis; hemorrhagic infarction of the lower lobe of the right lung; anasarca of the scrotum and lower extremities; general anæmia.

Duration of illness about three-quarters of a year. Swelling of the feet, general weakness, a sense of oppression in the chest—these were the leading symptoms observed by the patient, who confessed to the habit of drinking beer to excess. Urine very clear; specific gravity, 1.008; 4.5 per cent of albumin present. Under the microscope numerous leucocytes, kidney epithelial cells, granular casts, and triple phosphates were seen. There was also retinitis albuminurica, and toward the last the amount of albumin in the urine increased to seven per cent. (No. 136, 1894.)

Tab. 4.
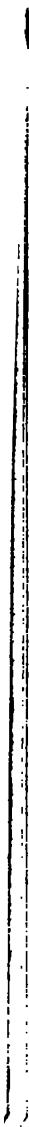
Explanation of Plate 6.

EMBOLIC (BENIGN) INFARCTION OF THE KIDNEY, COM-
BINED WITH NECROSIS OF THE TISSUES.

In the immediate vicinity of the pelvis of the kidney
is a cloudy-yellow, sharply limited pocket or area of
necrosed tissues, about the size of a hazelnut. This
focus of disease extends through all the intervening pa-
renchyma to the very surface of the kidney, its longer
axis running in a direction at right angles to this sur-
face. The remaining tissues of the organ possess a dirty
reddish-brown color, while the mucous membrane of the
pelvis is of a dirty grayish-green hue (this discoloration
being a post-mortem effect). The corresponding branch
of the renal artery was found to be plugged with a be-
nign, non-infectious embolus.

Tab. 6.

·Tab. 7.
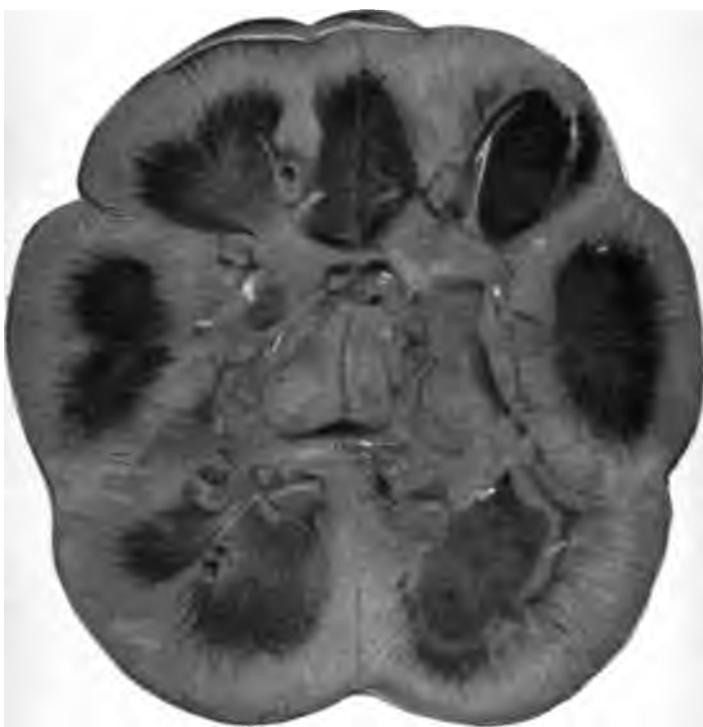
Explanation of Plate 7.

ACUTE TOXIC PARENCHYMATOUS NEPHRITIS, IN A CASE
OF BICHLORIDE-OF-MERCURY POISONING.

The kidney, taken from a girl nine years old, is
greatly enlarged; the capsule is easily removable; the
surface of the organ is smooth; its tissues are soft and
easily friable. The cortical substance is much broader
than normal and of a pale grayish-white color; the normal
markings, as seen on the cut surface, are indistinct, as if
they had been washed out. The pyramids are of a dark
bluish-red color, and somewhat swollen. The following
changes are recognizable under the microscope: Cloudy
swelling of the epithelial cells, partial necrosis of these
elements, and calcareous infarctions.

Three and a half days before her death the patient,
while in an excited condition, had swallowed a bichloride
tablet (1 gm.). Her intention, it was believed, was to
commit suicide. The greater part of the poison was
soon ejected from the stomach. Complete anuria. At
the post-mortem examination there was found, in addi-
tion to the marked nephritis, a catarrhal inflammation of
the intestines of moderate severity. *The mucous mem-
brane of the stomach was seemingly not affected.* Sub-
pleural ecchymoses over both lungs and hypostatic hy-
peræmia of the lower lobes. (No. 297, 1894.)

(This case of intestinal bichloride-of-mercury poison-
ing is described in greater detail in the dissertation of
Ralph Dürig, entitled "Ueber einen Fall von intestinaler
Sublimat-Intoxikation," Munich, 1896.)

Explanation of Plate 8.

SUBACUTE PARENCHYMATOUS AND INTERSTITIAL NEPHRITIS—"LARGE WHITE KIDNEY."

The kidney is enlarged to nearly double its normal size (its weight being 255 gm., instead of 140–150 gm.; length, 12.5 cm.; breadth, 7 cm.). The capsule was removed with ease, and the underlying surface of the organ was found to be smooth; the stellate arrangement of the veins was plainly recognizable. The consistence of the kidney was markedly diminished; its tissues were of a somewhat washed-out, grayish color (the reddish hue is to be ascribed to the deepening of color which takes place after death). When the kidney was cut open it could be seen that the cortex was broader than normal (1 cm.), and of a grayish color, with some admixture of red. The striations, which should normally be easy to recognize, were either effaced altogether or were only indistinctly marked in certain spots. A minimal quantity of blood was contained in the organ. A fairly abundant, opaque, and somewhat thickish juice was obtained when the edge of the knife was passed lightly over the surface of the section. The pyramidal substance was of a slightly darker color—a rather pale red—and the striations were plainly marked.

In the patient, who was nineteen years of age, the following pathological changes were found in other organs: Moderate hypertrophy and dilatation of both ventricles of the heart (partly of renal, partly of alcoholic origin); extensive, confluent, catarrhal pneumonia of both sides; sero-fibrinous pleurisy on the right side; moderate general dropsy. (No. 602, 1894.)

Tab. 8.

Tab. 9.

a

b

Explanation of Plate 9.

ACUTE PARENCHYMATOUS NEPHRITIS (STAGE OF FATTY DEGENERATION). THE NEPHRITIS OF PREGNANCY.

FIG. *a.*—Capsule very easily removed. Surface of kidney smooth and glistening. Color a pale grayish-yellow. Parenchyma easily friable. Boundary line between cortex and medulla sharply defined. The cortex, which is of a grayish-yellow color, contains but little blood, and the striations are not easily distinguishable. When the edge of the knife is drawn lightly over the cut surface of the organ an opaque juice is obtained in abundance. The pyramids are rendered conspicuous by their livid red color. When pressed upon, the papillæ give escape to an opaque, rather thick juice. The ureters are not dilated. Under the microscope it is seen that fatty degeneration of the epithelial elements of the urinary canaliculi is quite uniformly distributed throughout the organ.

Among the other pathological alterations which were found in this woman, who was forty-three years old and who had been delivered of her first child, by means of forceps, forty-six hours before her death (without convulsions), the following should be mentioned: A moderate degree of general dropsy; œdema of the lungs; commencing hypostatic pneumonia of both lower lobes; meningeal apoplexy extending over the left cerebral hemisphere and the right corpus striatum; multiple subserous myomata of the uterus. (No. 350, 1895.)

URIC-ACID INFARCTION OF THE KIDNEY IN A NEW-BORN CHILD.

FIG. *b.*—In the pyramids of the kidney, which displays a general icteric discoloration, uric-acid concretions of a golden-yellow color may be seen in the form of radiating striations.

Explanation of Plate 10.

Cystic Kidney.

Fig. *a.*—The external capsule contains very little fat, and the fibrous capsule can be separated from the organ itself only with difficulty. Both kidneys are very much reduced in size, and on their outer surfaces may be seen numerous transparent and thin-walled cysts of various sizes (from that of a hempseed to that of a cherry). Their contents are in some instances a clear, amber-colored fluid, in others a dark reddish fluid. In a section of the organ it can be seen that all through the parenchyma, which is in a high degree atrophic, there are numerous rather small cysts. At the hilus there is an abundant development of fat (*ex vacuo*).

In the patient—a man seventy-two years old and highly emaciated—the following lesions were also found: Brown atrophy of the heart, pulmonary emphysema, and extensive calcification of the retroperitoneal and mesenteric lymph glands (healed tuberculosis). (No. 445, 1895.)

Coral-Shaped Calculus from the Pelvis of the Kidney.

Fig. *b.*—The specimen was taken from a man, sixty-two years of age, who had died of purulent ascending pyelo-nephritis. A similar calculus was found in the other kidney. Both kidneys enlarged; cysts scattered everywhere throughout the tissues, which are much atrophied; medulla and cortex greatly narrowed; papillæ shortened; weight of both kidneys reduced to one-half of the normal weight; pelvis of both kidneys dilated; and mucous membrane thickened and covered with an opaque purulent fluid. Both ureters markedly dilated, and their lining mucous membrane swollen, discolored (a bluish-gray), and smeared with a thickish pus. In the urinary bladder there was a little fluid of a greenish color and somewhat resembling pus; and besides this there were two stones about the size of hen's eggs, shaped

Tab. 10.

a

b

Tab. 11.
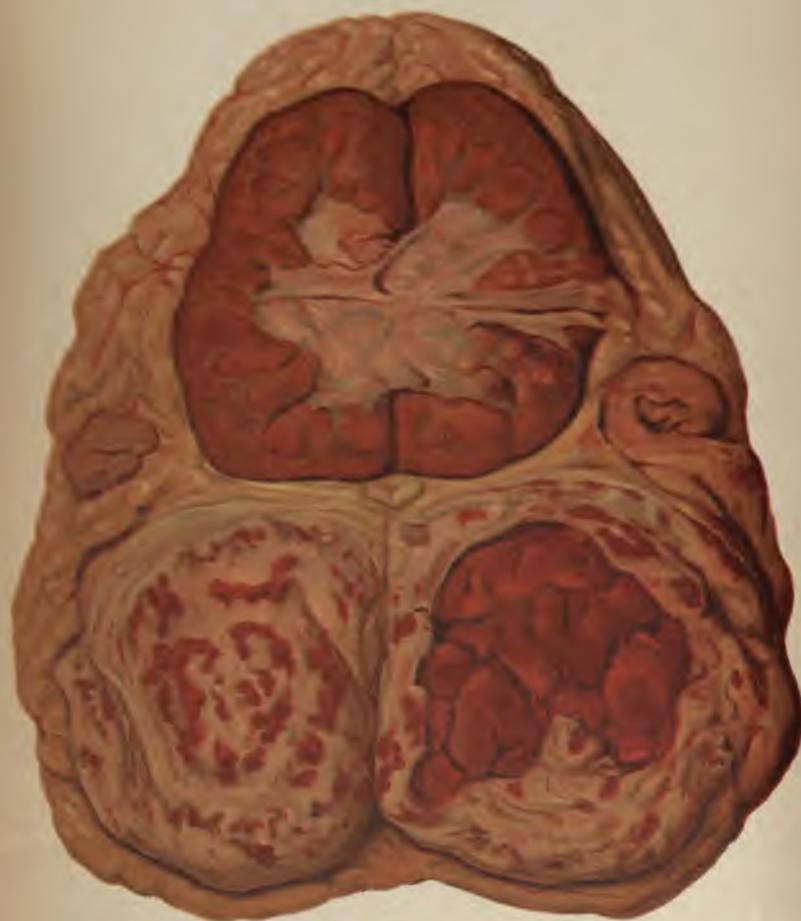
like breakfast rolls, of a clear grayish-yellow color, and lying side by side. The surface of these stones was covered with thorn-like projections. Among the other lesions found in this body were the following: Commencing uræmic enteritis; obsolete tuberculous disease at the apices of both lungs; emphysema of the lungs; brown atrophy of the heart. The body generally was much emaciated. (No. 719, 1895.)

Explanation of Plate 11.

Sarcoma of the Kidney.

(Reduced one-half in size.)

When the abdominal cavity was opened there was found, in the place of the kidney, a tensely elastic, fluctuating tumor, as large as the head of a child. This large mass, when cut into, proved to be composed partly of the kidney, and partly of a tumor (the size of two fists) which was attached to its lower border, and which was composed of spongy, whitish-yellow and orange-colored masses of tissue. In the centre of the tumor there was a cyst as large as a man's fist, and provided with a tough envelope of fibrous tissue. The kidney itself was separated from the tumor at almost all points, by sharply defined limits; but near the middle of the organ a point was visible on the surface where the tissues of the tumor were forcing their way into the kidney. Aside from this the renal parenchyma was somewhat atrophic and the pelvis dilated. As to the tumor, it is evident that it must have developed from some point on the surface of the kidney, presumably from some misplaced remnants of the suprareneal body which had found their way into the outermost portions of the cortex of the kidney, underneath the capsule, and had lain there harmlessly for a considerable length of time.

As causes of the death of this woman, who was fifty-five years old, may be mentioned the following: Septicæmia, originating in a phlegmon of the lower part of the right leg; emphysema of the lungs; and dilatation of the right ventricle of the heart. (No. 584, 1894.)

Explanation of Plate 12.

The kidney is very much enlarged, in some instances to nearly three times its normal size. Weight, in this particular case, 370 gm. (the normal weight, as the patient was very much emaciated, would be about 130 gm.). When the organ was cut open a thin, purulent, grayish-yellow fluid escaped from the dilated pelvis. The outer surface of the kidney is somewhat uneven through the presence of a number of low hummocks. The parenchyma, which is pale and saturated with moisture, presents everywhere throughout its substance irregularly shaped, and in some spots confluent, nodules and larger deposits of cheesy material. The pelvis is dilated in an irregular manner, there being at some points cavern-like diverticula which extend far into the pyramidal substance. Indeed, at one spot, the diverticulum—owing to the breaking down of the parenchyma—has pushed its way up to the very capsule of the kidney. The pale-yellow walls of these diverticula, which are filled with cheesy nodules, are at all points sharply separated from the neighboring tissues. To the left and somewhat below the middle line, in the picture, it will be seen how this cheesy infiltration of the pelvic mucous membrane extends down into the ureter itself. As a matter of fact, the tuberculous disease of the right kidney and the right ureter had extended so far as to involve the neck of the urinary bladder, the prostate, the seminal vesicles, one of the seminiferous canaliculi, and the corresponding testicle (genito-urinary tuberculosis; compare Plate 15).

The patient from whom this specimen was taken, and who was twenty-five years of age, died from an acute terminal basilar meningitis of a tuberculous nature, and was also affected at the same time with chronic tuberculous disease of the larynx, lungs, and intestines. (No. 11, 1895.)

Tab. 12.
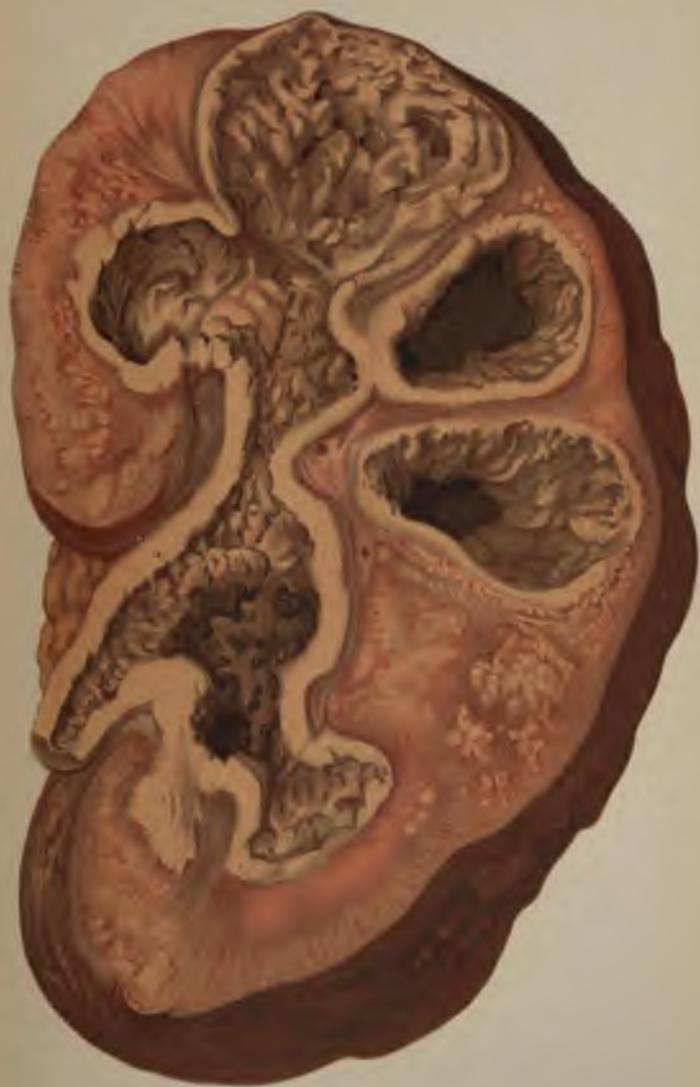
Lith.Anst.v.F.Reichhold, München.

Tab. 13.

Explanation of Plate 13.

PURULENT CYSTITIS, ASSOCIATED WITH NECROSIS OF
THE TISSUES; THE FORMATION OF DIVERTICULA OF
THE URINARY BLADDER; CICATRICIAL NARROWING
OF THE URETHRA, FALSE PASSAGES, PURULENT AND
GANGRENOUS PERI- AND PARA-URETHRITIS.

(Two-thirds natural size.)

The mucous membrane of the hypertrophied bladder
is irregularly reddened, very much swollen, and covered
with striated and spotted membranous masses. The
urine itself is discolored and opaque, contains pus and
mucus, and smells badly. Upon the left-hand side may
be seen a diverticulum, which has been laid open and
which communicates with the cavity of the bladder by
a narrow opening through which the probe, seen in the
picture, has been passed. As the cause and starting-
point of the purulent diphtheroid cystitis an old cicatri-
cial stricture may be pointed out in the membranous
portion of the urethra. In the neighborhood of this
stricture the soft parts show a number of centres of puru-
lent inflammation associated with the development of
gangrene. The pus in these centres is discolored and
has a foul odor, indicating that urine has begun to infil-
trate the parts. A few of the abscesses communicate
with the lumen of the urethra by means of fistulous
passages.

Explanation of Plate 14.

MULTIPLE TAR AND PARAFFIN WORKERS' CANCER OF
THE SCROTUM.   (After Volkmann.)

Several roundish cancerous outgrowths upon the sur-
face of the scrotum.   The smaller ones have elevated
wall-like margins, while the larger one (close alongside
the glans penis) presents small nodules and is fissured.
On the upper surface of the penis is seated a horny
cancerous growth about as large as a bean.

The patient, a man about forty-nine years of age,
showed evidences of the disease after he had worked for
eleven years in tar and paraffin manufactories.   Cured
by operation.

Tab. 14.

Tab. 15.

Explanation of Plate 15.

In the enlarged testicle may be seen two cheesy foci of considerable size. A broad bridge of parenchyma, which is also undergoing cheesy degeneration, is the only barrier between the two centres of softening. The larger of these has begun to break down, while in the smaller one the change has already advanced far enough to establish, at one end, the conditions of a cavity filled with fluid. These areas of disease are rather sharply separated from the surrounding healthy parts by walls of a tissue which is of an opaque yellowish color, easily broken down, and yet of fairly firm consistence. In addition to these lesions evidences of cheesy tuberculosis were found in the corresponding spermatic cord, in the seminal vesicles, in the prostate, in the neck of the urinary bladder, and in the right ureter, as well as in the pelvis and surrounding parenchyma of the right kidney (see Plate 12). The case, therefore, was one of *genito-urinary tuberculosis*, which doubtless began in the right kidney and spread downward from there to the different organs—which have been enumerated above—of the urinary and sexual apparatuses. The patient, a young man about twenty-five years of age, was very much emaciated. The causes of his death were: Acute tuberculous basilar meningitis, of which no symptoms had manifested themselves during his lifetime; chronic tuberculosis of the lungs; and ulcerating tuberculosis of the larynx and intestines. (No. 11, 1895.)

Explanation of Plate 16.

The uterus is as large as a man's fist, its walls measuring 2 cm. in thickness; its cavity, which is dilated, contains only scanty quantities of a dirty-grayish fluid mixed with altered blood. The mucous membrane displays shallow folds and small nodules, is discolored, and is covered in many places with superficial eschars. The os uteri is indented at several points. The vaginal mucous membrane presents an opaque and velvety appearance.

In this patient—a woman thirty-five years old, who had died, twenty-four days after the birth of her child, with symptoms which pointed to a severe *puerperal septicopyæmia*—numerous pathological alterations were found, viz., ichorous peri- and para-metritis; septic purulent thrombo-phlebitis of the left external iliac vein, of the common iliac vein, of the left femoral vein, and of the left vena saphena magna; anasarca of the left lower extremity (phlegmasia alba dolens); embolic plugging of the branches of the pulmonary arteries supplying both upper lobes of the lungs; and, finally, a metastatic ulcerous and thrombotic acute endocarditis of the mitral valve, with secondary embolic and gangrenous infarctions in the spleen and left kidney. Great septic enlargement of the spleen, this organ being nearly four times as large as it should be. On both nates extensive bedsores were found, the soft parts in these places having broken down into an ichorous pus. (No. 90, 1895.)

In the present case, which represents a combination of septico-pyæmia with ischorrhæmia, it is a circumstance worthy of remark that the veins and lymphatics of the uterus and its adnexa manifested—at least macroscopically—no characteristic alterations; and, further, that even the primary septic process in the endometrium was already, at the time when death occurred, in process of subsiding.

Tab. 16.

Explanation of Plate 17.

CARCINOMA OF THE UTERUS.

FIG. 1.—*Carcinoma of the Body of the Uterus.*—The uterine mucous membrane is covered with knobbed, soft, easily broken down masses; below, and to the right, a rather large, cauliflower-shaped nodule, which has been laid open with the knife, displays a grayish-white cut surface; the cavity of the uterus is dilated.

FIG. 2.—*Carcinoma of the Cervical Mucous Membrane of the Uterus.* (After Schaeffer).—In the thickened mucous membrane of the cervix quite a large number of smaller nodules, in size from that of a pin's head to that of a hempseed, may be seen embedded in its substance. At one spot ulceration has already taken place. On the surface of the section, particularly on the right side, one can see how the cancerous growth has already invaded the deeper parts of the uterus and has involved the muscular structure.

Fig.1.

Fig.2.

Explanation of Plate 17.

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Explanation of Plate 18.

Tuberculosis of the Uterus, Vagina, Fallopian Tubes, and Left Ovary.

The cavity of the uterus contains a yellowish, greasy-looking substance, which evidently represents uterine tissue in a state of cheesy necrosis. The mucous membrane, throughout its entire extent, is converted into a friable, opaque-yellow, cheesy material. The necrotic process extends, at numerous places, into the deeper layers of the muscular coat. The left Fallopian tube is represented by a tough, thickened cord; its lumen is dilated, and the mucous membrane which lines it, like that of the uterus, is in a condition of diffuse cheesy necrosis. The right tube is in much the same condition. The left ovary is also converted into a crumbling, cheesy mass. Upon the posterior upper wall of the vagina there are several shallow ulcers, coated with an opaque-yellow material, and separated from the surrounding tissues by sharply defined limits. Douglas' space is obliterated (adhesive pelveo-peritonitis). Upon the peritoneal covering of the uterus and its appendages there are numerous tuberculous eruptions, some of them arranged after the manner of a string of pearls.

In addition to the lesions described above, there were found in this patient—a woman, fifty-four years of age—the following pathological conditions: Ulcerating tuberculosis of the upper portions of the colon; secondary cheesy tuberculosis of the mesenteric and retroperitoneal lymph glands; and also tuberculosis of the abdominal peritoneum, with slight inflammatory alterations (subchronic tuberculous peritonitis). Finally, in the upper lobe of the left lung there were evidences of localized apex-tuberculosis of older date, together with

Tab. 18.

.

Tab. 19.

Lith.Anst.v.F.Reichhold, München.

a certain amount of subacute trouble of more recent origin.

It is not clear whether the genital tuberculosis noted in this case is to be considered as the primary disease or merely as a secondary development. In the latter case —which is the more likely of the two to be the correct one—it may be assumed that the infectious process spread from the mucous membrane of the colon to the lymph glands of the abdominal cavity, to the peritoneum, and thence to the Fallopian tubes and the mucous membrane of the uterus and vagina. (No. 383, 1895.)

Explanation of Plate 19.

POLYPOID FIBROMA OF THE UTERINE MUCOUS MEMBRANE (MUCOUS POLYP).

In the cavity of the uterus, which is moderately enlarged, is a tumor about half the size of a hen's egg. It grows from the upper and posterior wall. It is of a dark red color and soft consistence. Its surface is somewhat knobbed, and it is attached by a broad base to the wall of the uterus.

The patient, a woman forty-six years of age, died from tuberculosis of the lungs. (No. 387, 1895.)

Explanation of Plate 20.

In the walls of the uterus there is quite a large num-
ber of myomata, varying in size from that of a pea to
that of a walnut.   While a few of them are located deep
down in the muscular layer, the majority lie near the
surface, just beneath the serous covering.   The tissues
covering the fundus and body of the uterus have, for
the most part, been obliterated by the tumors, but the
cervical portion of the organ (which has been laid open
by a longitudinal incision) is still fairly well preserved.
The grayish translucent sac, which lies to the left of the
disfigured uterus, and whose walls display a somewhat
rich development of blood-vessels, is nothing more or
less than the enormously dilated left Fallopian tube,
which is filled with a serous fluid (hydrosalpinx), and
whose orifices have been obliterated.

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Tab. 20.

Tab. 21
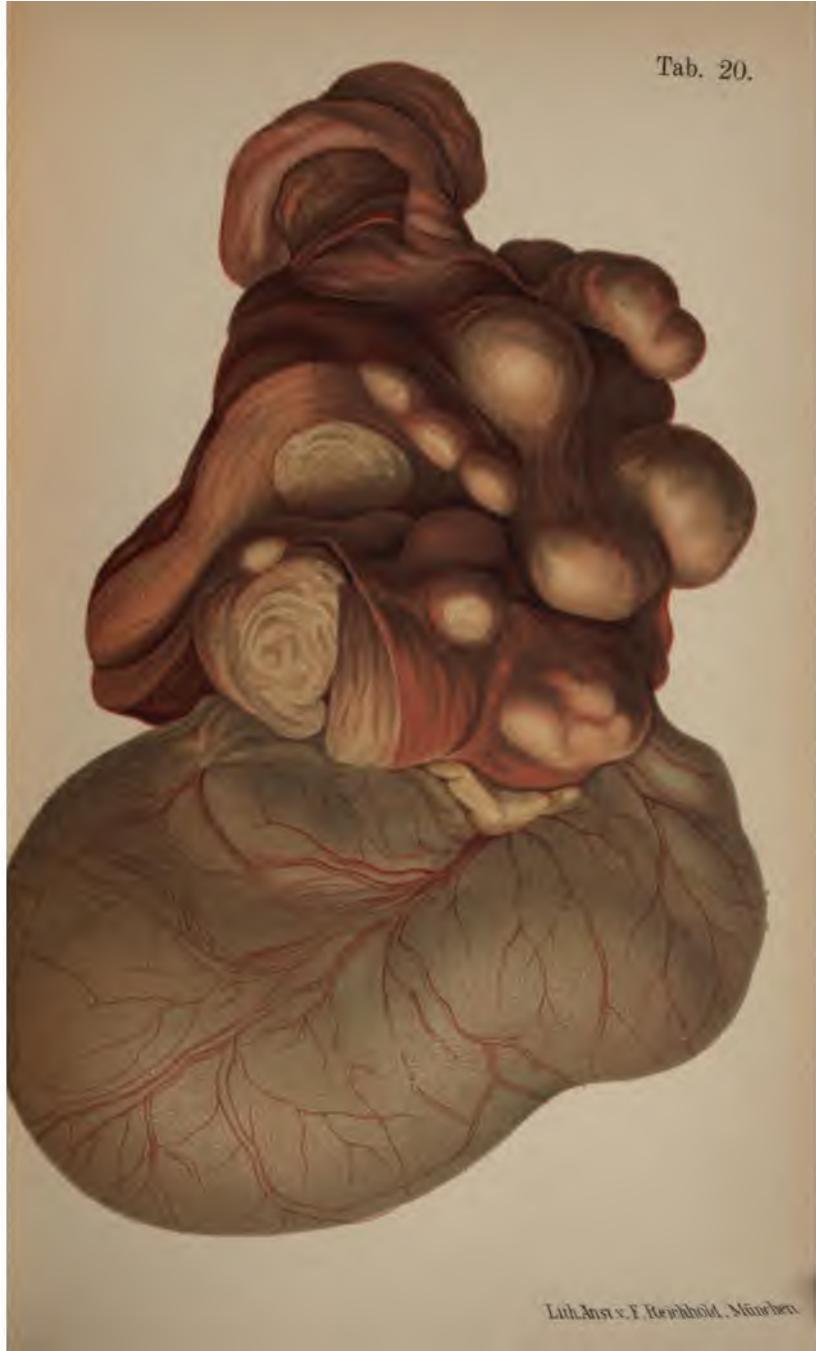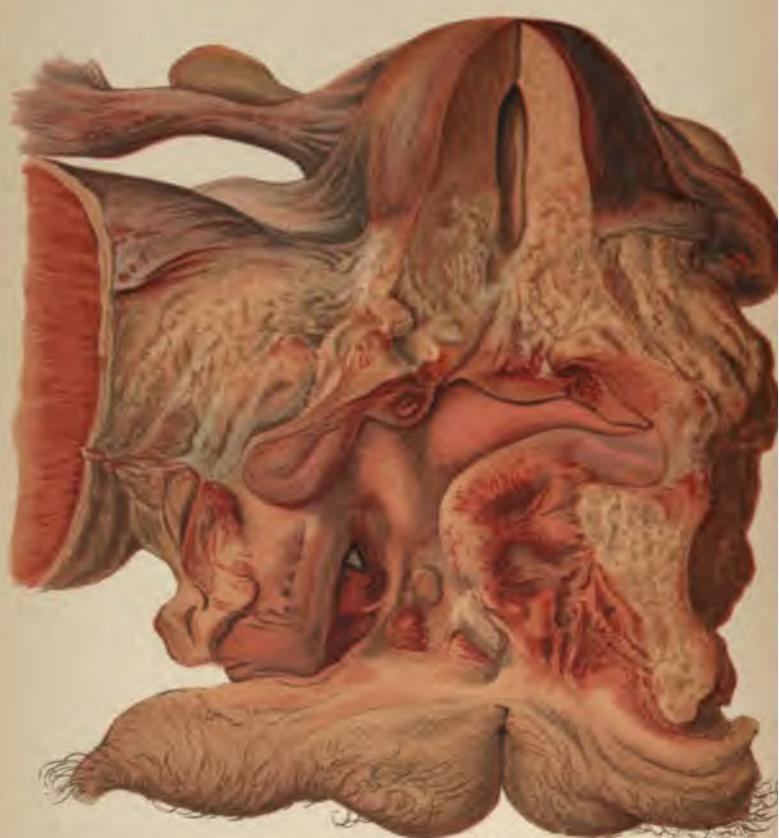
Explanation of Plate 21.

CARCINOMA OF THE CERVIX UTERI, WITH BEGINNING
    INVOLVEMENT OF THE VAGINA AND POSTERIOR
    WALL OF THE BLADDER.

(Two-thirds natural size.)

The cervical portion of the uterus is, throughout its entire extent, infiltrated with cancer elements; these cancerous portions being distinguished from the healthy tissues by their opaque-yellowish and whitish color and by their diminished consistence. Below and toward the front the cancerous infiltration is advancing into the vault of the vagina and into its anterior wall, from which region, in turn, it is encroaching upon the posterior wall of the bladder. The subperitoneal connective tissue in the vicinity of the vagina and bladder, and the continuation of this downward as far as to the vulva, are also full of disseminated cancer masses.

Explanation of Plate 22.

RETRO-UTERINE HÆMATOCELE IN CONNECTION WITH
AN EXTRA-UTERINE FŒTAL SAC.

Among the masses of clotted blood which had been deposited between the Fallopian tube and the fœtal sac, a one-month-old embryo was found.

Tab. 22.

Tab. 23.

Explanation of Plate 23.

ADHESIVE PELVEO - PERITONITIS, PERI - OÖPHORITIS, PERISALFINGITIS, AND PYOSALPINX ON THE RIGHT SIDE. (After Schaeffer.)

Douglas' space seen from behind and above. Numerous pseudo-membranes, in the form of bands and more delicate cobweb-like threads, connect the posterior surface of the uterus with the neighboring organs, viz., the Fallopian tubes, the ovaries, and the sigmoid flexure of the large intestine, a segment of which may be seen in the picture. The left Fallopian tube is bent at several points; the outer surface of the right tube is inflamed and red, and its internal surface is in a condition of purulent inflammation. By reason of this and of the closure of the abdominal orifice, this tube is converted into a pyosalpinx, which is subdivided into well-rounded sections.

Explanation of Plate 24.

Syphilitic Pemphigus of Newly Born Children, in Congenital Syphilis. Bullous Syphilide of the Skin.

(One-third natural size.)

The skin is covered with numerous quite large, bladder-like structures which are filled with pus, and which are very distinct and numerous upon the soles of the feet. These have developed out of livid or brownish-colored spots, which rapidly become converted into pustules and bladders filled with pus. After the latter have ruptured and the pus has been poured out, the formation of ulcers and scabs follows. Through the confluence of several bladders extensive secreting ulcers, with infiltrated bases, may develop. This skin syphilide seems to manifest itself with special frequency on the palms of the hands and the soles of the feet. Children affected with hereditary syphilis are born with the exanthem upon their bodies, or it breaks out shortly after birth. Recovery from the disease is a possibility. In many cases these superficial sores may develop into deep ulcerations and gangrenous sloughs.

Gummous products are often found in the internal organs. Osteo-chondritis of the margins of the epiphyses (Wegner) and still other pathological alterations (e.g., disfigurement of the upper incisor teeth, and keratitis parenchymatosa) are also encountered.

Tab. 24.

Tab. 25.

Explanation of Plate 25.

MULTIPLE FIBROMATA (NEURO-FIBROMATA) OF THE SKIN.

Over the skin of the entire body is scattered a very large number (several thousands) of fibromata, which vary in size from that of a pin's head to that of an English walnut. These tumors are composed of fibrillated connective tissue; they originate in the deeper layers of the cutis (the stratum reticulare) and appear to spring more directly from the connective-tissue sheaths of the nerves which are distributed throughout the cutis. (They are also known as false neuromata or pseudo-neuromata.) The nerve fibres pass almost unchanged through the tumors, and no new growth of nerve tissue has ever been demonstrated in these fibromata.

Explanation of Plate 26.

The lateral ventricles are very markedly dilated, and filled with a clear watery fluid. The ependyma is somewhat opaque and thickened. (The dark coloring of the walls of the ventricles did not exist in the original preparation, from which the drawing was made; it is due to the fault of the artist, who made his shading too dark.) The secondary disappearance of the cerebral substance may be seen with special distinctness in the white matter of the cerebral hemispheres, while, on the other hand, the gray cortical matter appears to be less narrowed.

Tab. 26.
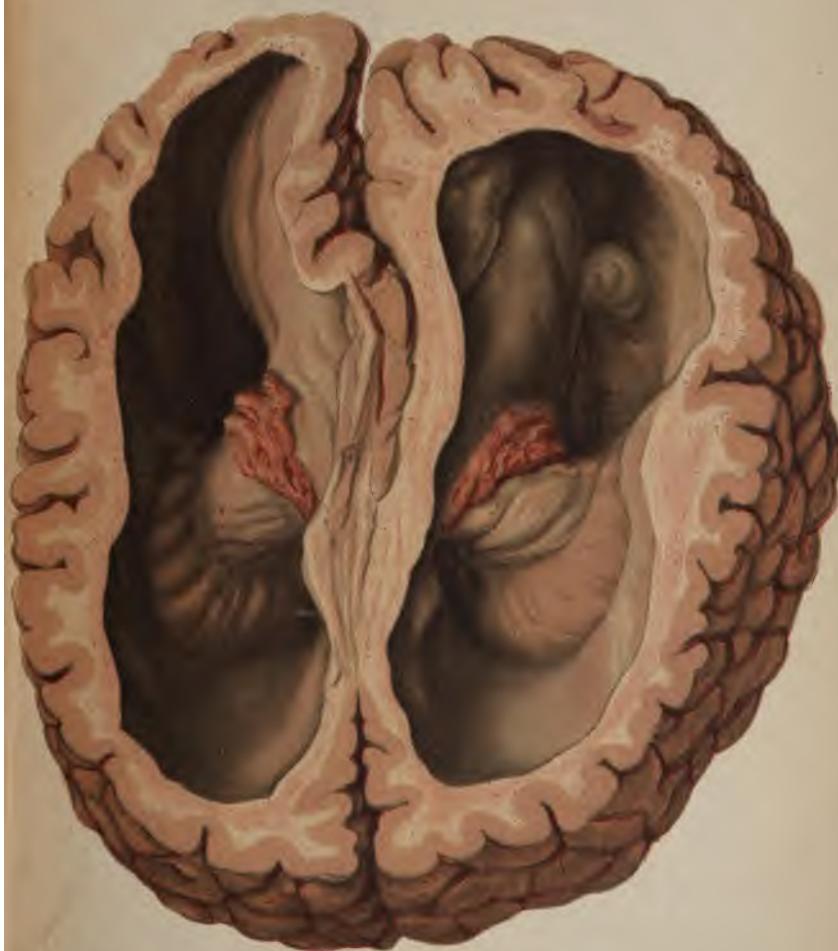
Tab. 27.

Explanation of Plate 27.

CHRONIC CONGENITAL HYDROCEPHALUS.

The case under consideration is that of a female child, fourteen months old. At the post-mortem examination the head looked as if it had been blown up like a balloon. The cerebral part of the skull was very much larger than the facial part. The frontal and parietal prominences stood out conspicuously. All the sutures and fontanelles gaped widely open. The large fontanelle measured 12 cm. in length, and 5 cm. in breadth. The circumference of the skull measured 55 cm. There were extensive defects in the vault of the cranium. After the latter had been removed, and the enormously distended ventricles had been emptied of their contents (about 1,400 c.c. of fluid as clear as water), the cerebral hemispheres presented the appearance of two collapsed bladders. This shell of cerebral substance measured only 10 or 11 mm. in thickness. (The condition may properly be termed secondary pressure atrophy of the cerebral hemispheres.)

The other pathological conditions found in this greatly emaciated child (which weighed only 6.5 kgm.) were the following: Tuberculosis of the peribronchial and mediastinal lymph glands; confluent lobular pneumonia on the right side; fatty liver; and rhachitis. (No. 10, 1895.)

Explanation of Plate 28.

(Lumbar Portion of the Spinal Cord.)

When the dural sac was opened, the soft membranes presented a cloudy appearance; they had lost their natural degree of transparency, and displayed in some places red spots. The meshes of the arachnoideus and pia were filled with a grayish-white (in some places yellowish and yellowish-white) exudation which varied in character; being gelatinous at some points, sero-purulent at others, and distinctly purulent at still others. A similar exudation, but of varying thickness, was found throughout the middle and upper portions of the spinal cord, along the base of the brain, and extending up over the side of this organ as far as to the convexity of the left hemisphere. It was also found that the infectious purulent inflammation had involved the plexus of veins and the walls of the lateral ventricles; the latter cavities being filled with a sero-purulent fluid (pyocephalus internus). The duration of the entire illness, which throughout pursued the typical course of an attack of epidemic cerebro-spinal meningitis, was fourteen days. The patient, a woman thirty-two years of age, died in convulsions (clonic spasms). The other pathological alterations found in this case were: Anæmia of the component tissues of the brain and spinal cord; hypostatic congestion of both lower lobes of the lungs; and cyanosis of the kidneys. (No. 272, 1894.)

Tab. 28.

Explanation of Plate 30.

Directly behind the left side of the frontal bone lies a
large mass of clotted blood, which has separated the dura
mater from the inner wall of the bone throughout a wide
extent. The hemorrhage came from the middle menin-
geal artery, which was torn at a point corresponding to a
traumatic fissure in the frontal bone. The surface of
the brain in the corresponding region displays a shallow
depression.

Tab. 30.
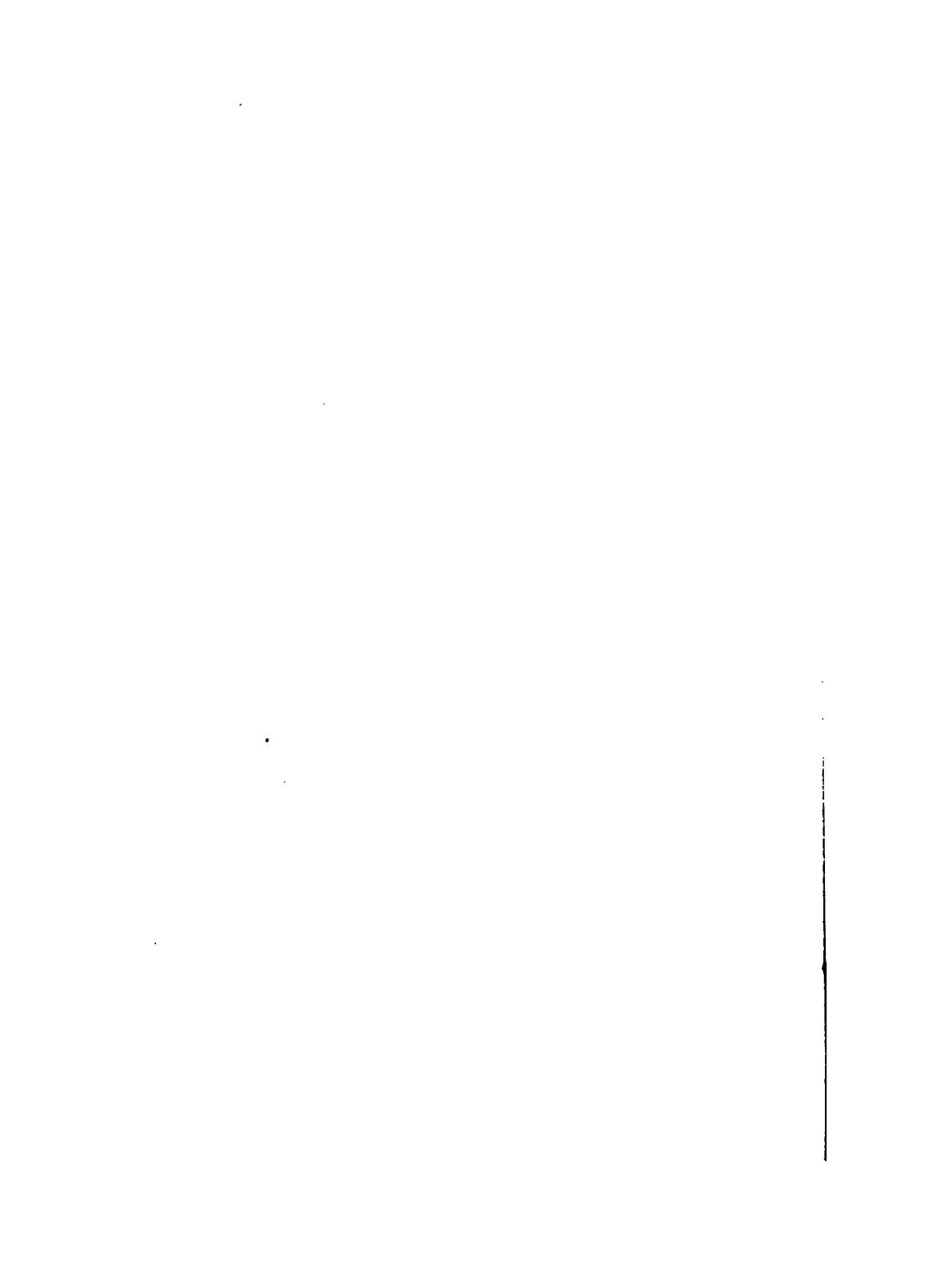
Tab. 31.

Explanation of Plate 31.

PACHYMENINGITIS INTERNA HÆMORRHAGICA. PACHY-
MENINGITIS VASCULOSA CHRONICA.

(Two-thirds natural size.)

The inner surface of the dura mater (right half) is covered with a pseudo-membrane which is colored a pale red in some parts, a dark red in others, and which clings firmly to the dura. On the left side there is presented an irregularly-shaped yellowish-green deposit which rests upon an older layer of organized exuded material of a dark red color and infiltrated with blood. The explanation of these conditions is this: An infectious fibrino-purulent inflammation has, in this case, been grafted upon an older proliferative process, a complication which is observed only in rare instances. The pseudo-membrane, which is applied quite uniformly over the inner surface of the dura mater, is composed of a fibrous connective tissue which in some parts is rich in cells and everywhere is liberally supplied with blood-vessels (pachymeningitis vasculosa). In many places throughout the layers of newly formed tissue there may be seen hemorrhagic exudations of older and more recent dates. It is safe to assume that these hemorrhages have come from the imperfectly developed new blood-vessels, through a process of diapedesis.

Explanation of Plate 32.

In the right cerebral hemisphere, at a point corresponding to the location of the nucleus lentiformis, and extending from this outward as far as to the capsula interna, is an irregularly shaped cavity, somewhat larger than an English walnut, which is filled with dark, reddish-brown masses of clotted blood. Its walls in some places show a rusty red discoloration. Some bloody serum is present in the right lateral ventricle. The rest of the cerebral parenchyma is tough, the brain as a whole being atrophic (it weighs 1,220 gm.). The large arteries at the base of the brain are in a condition of marked atheromatous degeneration; the arteria pro fossa Sylvii is obliterated.

Among the other pathological conditions found in this woman, who was sixty-nine years of age, the following may be mentioned: Chronic interstitial nephritis and hypertrophy of the heart (both ventricles), which weighed 475 gm. (the normal weight being from 250 to 300 gm.). (No. 81, 1895.)

Tab. 32.

Tab. 33.

Explanation of Plate 33.

RED SOFTENING OF THE RIGHT CEREBRAL HEMISPHERE;
HEMORRHAGIC ENCEPHALO-MALACIA (eighteen days
old).

In the vicinity of the left temporal lobe and island of
Reil the brain substance felt very soft, even fluctuating,
like a bladder filled with fluid. When this portion was
cut into, by an incision which began at a point corre-
sponding to the left centrum semiovale and extended
from there down as far as the island and the cortex of
the temporal lobe, a cavity as large as a child's fist was
exposed to view. This cavity was filled with a rust-
colored, soft, greasy material, which consisted of
broken-down brain substance, and throughout which
numerous small collections of blood were scattered. The
white brain substance in the neighborhood of the cavity
was in an œdematous condition and lemon colored.
Toward the median line the area of softening extended
as far as to the ependyma of the left lateral ventricle,
and included the thalamus opticus and the nucleus lenti-
formis. The blood-vessels at the base of the brain
showed no disease of their walls; the arteria pro fossa
Sylvii, on the left side, was empty.

In this patient—a woman fifty-four years of age, who,
eighteen days previously, had had an apoplectic attack—
the following pathological alterations were found in
other parts of the body: Chronic endocarditis of all the
valves of the heart, with the exception of those placed
at the commencement of the pulmonary artery; recur-
ring verrucose endocarditis of the mitral valve, with
marked stenosis of the same; stenosis of the tricuspid
valve, and the presence of a ball-shaped thrombus in the
right auricle; multiple hemorrhagic infarctions in both

lungs; inhalation pneumonia of the right lower lobe; passive congestion of the different organs. In view of the fact that the patient had endocarditis of the cardiac valves (aortic and mitral) the diagnosis of embolism of the arteria pro fossa Sylvii was made during her lifetime. (No. 45, 1895.) However, as no embolic plugging of the larger portions of this vessel and no sclerosis of the cerebral arteries were found at the post-mortem examination, the idea of an embolic plugging of some of the finer ramifications of this artery, or of a hemorrhage due to passive congestion, must, in the present case, be given the preference.

Explanation of Plate 34.

PORENCEPHALIA.

On the under surface of the brain, on the left side, may be seen a long groove or pit-like depression, representing a deficiency of brain substance throughout this particular area. It measures 10 cm. in length, and between 3 and 4 cm. in breadth. It begins at the posterior end of the left occipital lobe, extends in a forward direction entirely across it, and then involves the posterior and median portions of the temporal lobe. This pit was filled with serum, and the soft membranes which covered it were somewhat opaque, of a light yellowish-brown color, slightly thickened, and very richly provided with blood-vessels.

In this particular case it is not unlikely that the picture presented is that of a secondary porencephalia, one which developed in childhood as a result of a hemorrhage from some blood-vessel in this region. (It might be termed a case of pseudo-porencephalia.)

Tab. 34

Tab. 35.
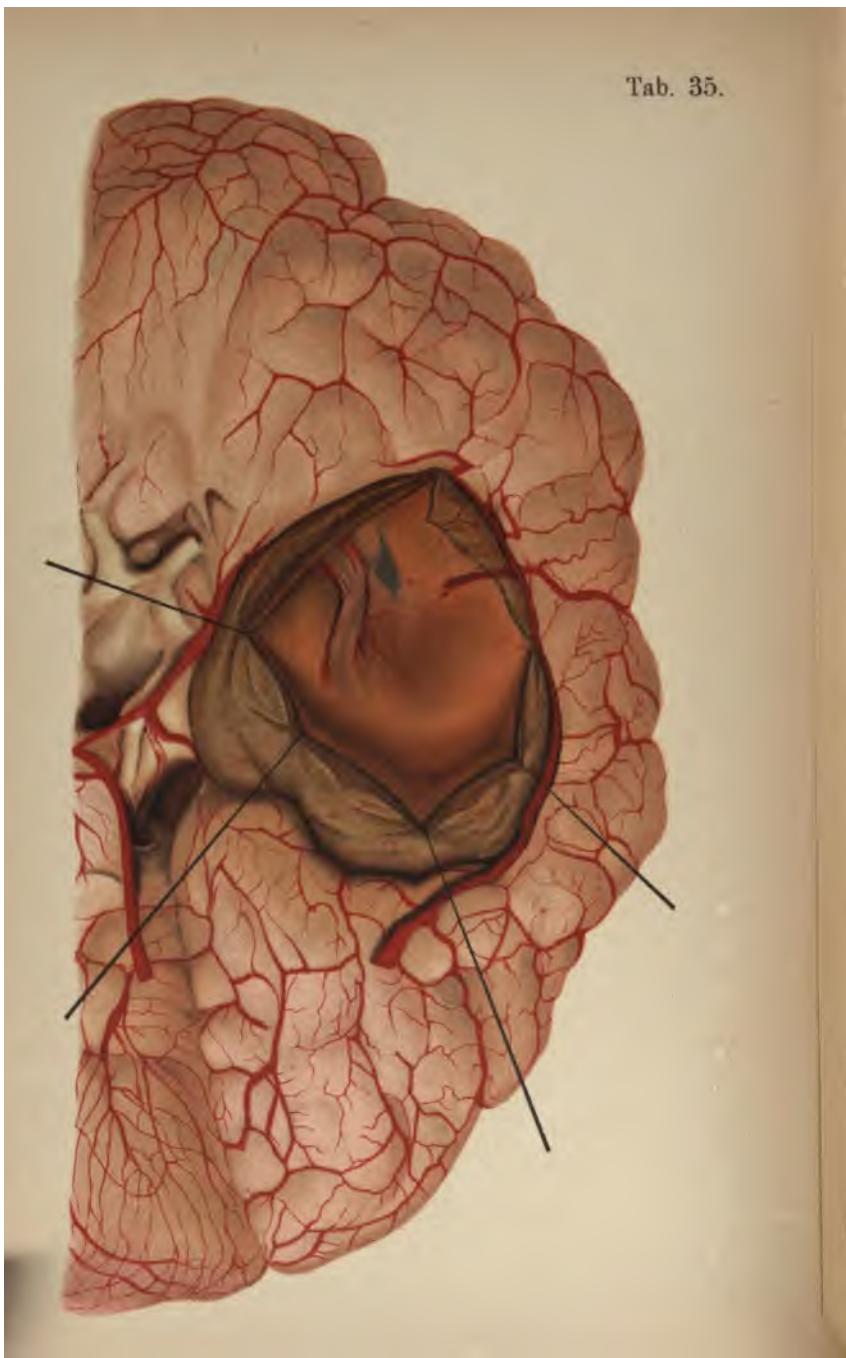
Explanation of Plate 35.

PORENCEPHALIA IN THE LEFT TEMPORAL LOBE, WITH
ENCYSTED MENINGEAL HÆMATOMA.

At the base of the brain, at a spot corresponding to
the left fossa Sylvii, there is a bladder, about as large
as a small apple, which fluctuates and is filled with fluid.
Toward the median line it extends as far as to the middle
of the chiasma; toward the outside it extends as far as
the margin of the base of the brain; and in a forward
direction it is limited by the frontal lobe. The color of
this bladder is in part a dirty yellowish-brown, in part
that of iron rust. Delicate blood-vessels are visible
upon its surface. In the region of the right fossa Sylvii
there is a smaller bladder, about as large as an English
walnut, which is filled with a clear serous fluid. The
margins of both these bladder-like structures were found
to be so intimately united with the neighboring cerebral
cortex that all our efforts to effect a separation proved
unavailing.

The cause of death in this patient—a girl four and
a half years old—was diphtheria of the larynx; and, in
addition, there were found: Double croupous pneumonia,
excessive general emaciation, anæmia, and rhachitis.
(No. 437, 1895.)

Explanation of Plate 36.

PRIMARY, MULTIPLE, CHRONIC TUBERCULOUS DISEASE
OF THE RIGHT CEREBELLUM.

The convolutions of the cerebrum were found to be flattened and the furrows obliterated. Irregularly shaped, tough, cheesy, opaque-yellow nodules were disseminated throughout the greater part of the right cerebellum; and in the immediate neighborhood of these nodules the brain substance appeared to be somewhat softened and of a grayish-red color. The tuberculous nature of the tumor-like foci was established both by the aid of the microscope and by bacteriological methods. All the ventricles of the brain were very much dilated and filled with a watery fluid (hydrocephalus internus chronicus). The ependyma was found to be somewhat macerated.

In this patient—a man forty-six years of age—the following lesions were found in other parts of the body: Emphysema of the lungs and hyperæmia of the abdominal viscera. No trace of tuberculous disease was found anywhere in the body outside of the cerebellum. (No. 319, 1895.)

Tab. 36.

Tab. 37.

a

b
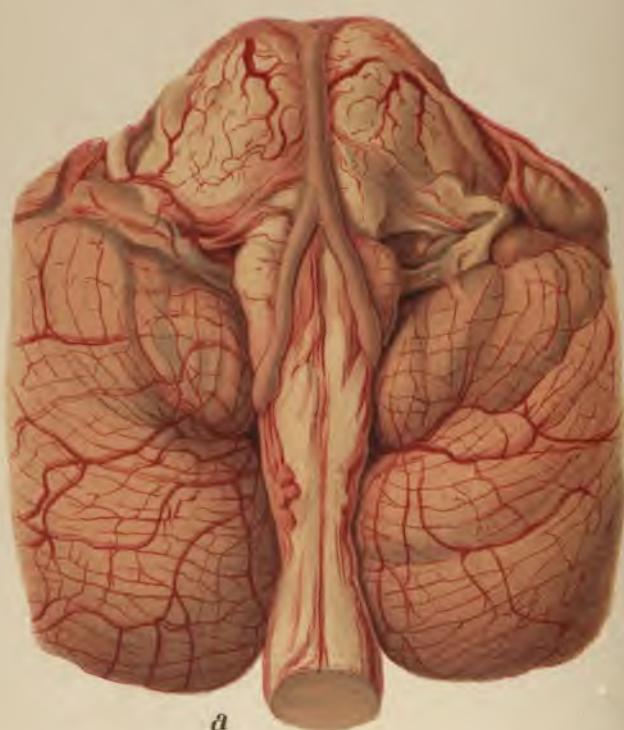
Explanation of Plate 37.

SPINAL-CORD LESIONS DUE TO TRAUMATISM (*a*) AND
TO POLIOMYELITIS CHRONICA (*b*).

(*a*) *Compression Myelitis in the upper Cervical Portion
of the Spinal Cord, in a Case of Spontaneous Frac-
ture of the Odontoid Process of the Axis, or Second
Cervical Vertebra.*

At a distance of 3.5 cm. from the pons the upper part of the
cervical portion of the cord presented a distinct narrowing, and,
immediately after these parts had been exposed to view, this
narrowed condition stood out still more conspicuously. The
substance of the spinal cord, at the point of contraction, was
found to be in a softer condition than were the neighboring
healthy parts. At this place also the vertebral canal was
markedly narrowed, and the spinal dura mater bulged out in a
backward direction and seemed to fluctuate. This gave the im-
pression, in the earlier part of the post-mortem examination,
that the narrowing of the canal was due to the presence of a
semisolid extradural tumor (sarcoma); but, upon further exam-
ination, it was ascertained that a spontaneous fracture had taken
place in the odontoid process of the carious second cervical ver-
tebra.

The patient, a boy sixteen years of age, was affected, during
his lifetime, with paralysis of all the extremities, and the diag-
nosis which had been made was that of a cervical spondylitis.
Among the pathological alterations found elsewhere in the body
were the following: Broncho-pneumonia of both lower lobes,
and of the right middle and upper lobes; subacute verrucose en-
docarditis of the mitral valve; general emaciation (weight of
body = 34 kgm.) and anæmia. (No. 168, 1895.) A more detailed
description of this case will be found in the dissertation of Martin
Lubinski, entitled: "Kasuistischer Beitrag zur Lehre von der
Kompressionsmyelitis," Munich, 1895.

(*b*) *Multiple Capillary Apoplexies in the Posterior
Columns of the Spinal Cord in Poliomyelitis
Chronica.*

Upon the transverse section of the spinal cord there can be seen
quite a large number of punctiform fresh hemorrhagic foci (capil-
lary apoplexies), which do not disappear when the edge of the
knife is drawn lightly over them. Similar multiple capillary
apoplexies were found in the parenchyma of the cerebrum.

As the cause of death of this patient—a young woman, nine-
teen years old—there was found an acute, generalized, miliary
tuberculosis of the lungs, liver, kidneys, and peritoneum; the
disease having probably originated in an ulcerating primary
tuberculosis of the intestines and a cheesy tuberculosis of the
mesenteric glands.

Explanation of Plate 38.

UNITED FRACTURE OF THE FEMUR, AS SEEN FROM TWO
SIDES. (After Helferich.)

The different forms of dislocation of the fractured ends
of the shaft are here combined. They are as follows:

1. The two broken fragments are displaced in such a
manner that their ends do not stand facing each other,
but lie side by side. This form is termed lateral dis-
location—dislocatio ad latus (see Fig. *a*, in the plate).

2. The two fragments are not only dislocated laterally,
but they are also displaced longitudinally in such a man-
ner that the femur as a whole appears to be shortened;
the fragments ride past each other. This form is termed
longitudinal dislocation with contraction—dislocatio ad
longitudinem cum contractione (see also Fig. *a*, in the
plate).

3. The fragments do not lie side by side in parallel
lines, but the respective axes of the two form somewhat
of an angle with each other. This form is termed axial
dislocation—dislocatio ad axin (see Fig. *b*, in the plate).

4. In this fourth form one further change is added to
the three just described, viz., one of the fragments has
undergone a certain amount of twisting around its long
axis. In Fig. *a* the upper fragment is seen in a correct
front view, while the lower fragment seems to be twisted
very decidedly inward. This form is termed peripheral
dislocation—dislocatio ad peripheriam.

Tab. 38.

a

b

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Tab. 39.

a

b

C

Explanation of Plate 39.

Complicated Fracture of the Thigh.

FIG. *a.*—The fractured ends of the bone, in consequence of a septic inflammation of the wound, became necrosed throughout their entire thickness.   After the lapse of months, the sequestra formed in this manner separated from the living bone. Finally, amputation was resorted to, inasmuch as there seemed to be no prospect that a consolidation of the parts would take place.   In Fig. *a* (the lower one of the three) the upper part of the lower fragment of the broken bone is shown in its entire thickness, with the sequestrum in place.   The latter presents the appearance of a macerated portion of bone, whose upper extremity shows the irregular edges of the line of fracture.   The lower part of the sequestrum is surrounded by newly produced bone substance, which is characterized by osteophytic deposits, specially numerous near the sequestrum, and presenting some resemblance to dripstones.   The uppermost of the three figures represents a section of the upper fragment of the broken bone. The external deposits of bone, the beginning of the process of absorption in the compact substance, and the obliteration of the medullary cavity by the development of spongy bone substance, all these things are well shown in the drawing.   The middle picture represents the sequestrum belonging to the upper fragment of bone.   Below may be seen the irregular edge corresponding to the line of fracture, while above there are tooth-like projections—the product of the inflammation which progressed slowly during the separation of the sequestrum from the living bone.

Healed Fracture of the Humerus (section).

FIG. *b.*—Slight axial dislocation.   At the point of fracture one can see how the compact ends of the fragments are united together by a scanty mass of callus, which also appears to be composed of a certain amount of compact substance.   The medullary cavity is open, and it is only at the spot where the fracture occurred that a certain amount of narrowing, due to the presence of a few lamellæ of spongy bone, can be seen. ·

Fracture of the Tibia which has Healed at an Angle (section).

FIG. *c.*—The fragments are very much displaced and they have become united by their sides.   The old compact cortical portion has assumed a more spongy character.   The two portions of the medullary cavity are separated from each other by the cortical portions of the two fragments and by the bone substance which has been deposited in large quantity between them.

Explanation of Plate 40.

RHACHITIS OF THE FEMUR AND ONE OF THE RIBS.

Rhachitis of the Lower End of the Femur.

FIG. *a.*—The lower end of this bone is very much swollen; it is more than half as large again as it should be. The normal, white, cartilaginous epiphysis is placed next to a bluish-colored zone of cartilage which is broader than normal and is in a state of active proliferation. Then, next to this, comes the layer of osteoid tissue which is provided with blood-vessels and medullary spaces, which are pushing their way into the cartilaginous zone; while, higher up still, may be seen the pale red and spotted ordinary osteoid tissue. Last of all comes the medullary cavity, surrounded by the thin and compact bony cortex.

Rhachitis of a Rib, at the Point where the Cartilaginous and Osseous Portions Unite.

FIG. *b.*—At the point of union between the bony end and the cartilage the rib is appreciably swollen (rickety rosary). There is excessive growth of cartilage with insufficient calcification. The zone of cartilage is increased in breadth, it has an abnormal bluish color, and the outline of its boundary is irregular. Through the incomplete substitution of medullary spaces for cartilage tissue, the latter may be said to have undergone a direct conversion into osteoid substance, as a result of which there is a certain amount of enlargement of the affected region.

Tab. 40.

a

b

Tab. 41.
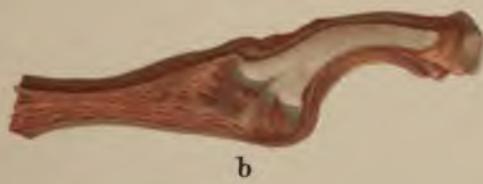
Explanation of Plate 41.

PELVIS AFFECTED WITH OSTEOMALACIA.

The lateral pressure exerted by the thigh bones has caused the symphysis to protrude forward like a beak, and has materially diminished the transverse diameter of the pelvis. The rami of the os pubis are curved to an unusual degree. Owing to the pressure exerted by the trunk, the os sacrum is pushed down to an abnormally low level. The internal surfaces of the ilia are also abnormally curved and lengthened laterally. The outlines of the upper entrance to the pelvis are like those of the figure of a heart in playing-cards.

The entire pelvis weighs less than it normally should, and in the fresh state it yields as if made of India rubber; it is also easy to cut.

Explanation of Plate 42.

LYMPHOID HYPERPLASIA OF THE BONE MARROW IN A
CASE OF SPLENIC, LYMPHATIC, AND MYELOGENOUS
PSEUDO-LEUKÆMIA.

The medullary cavity of the femur is filled with a partly bright red and partly dark red marrow, which looks like strawberry jam. In consequence of hyperplasia of its cellular elements, the originally whitish-yellow and fatty marrow has here been converted into a mass of reddish lymphoid tissue.

The patient from whom this specimen was taken, and who died of a profound anæmia, was thirty-two years of age and gracefully built. At the post-mortem examination various other pathological conditions were found, as, for example: Enormous enlargement of the spleen; lymphoid hyperplasia of the intrathoracic and retroperitoneal lymph glands; a high degree of general anæmia; a terminal sero-fibrinous pleuritis on the right side; and bronchiectasiæ in the upper lobe of the right lung. (No. 208, 1895.)

Tab. 42.

Tab. 43.

Explanation of Plate 43.

The bone, throughout its entire extent, is enlarged, and covered with an abundance of osteophytic growths (periostitis ossificans). Here and there the opening of a cloaca may be seen. In the lower part of the bone, throughout a length of several centimetres, a smooth sequestrum is visible.

Explanation of Plate 44.

CHRONIC OSSIFYING OSTEOMYELITIS OF THE TIBIA,
WITH PARTIAL NECROSIS OF THE BONE.

The medullary cavity is only at one or two spots, par-
ticularly in the middle part of the bone, still distinctly
recognizable. Everywhere else a spongy and in some
places sclerotic mass of bone has developed in its place.
Near both ends of the bone irregularly shaped cavities
may be seen. When the parts were in a fresh condition
these cavities were filled with pus, and the lower one
opened outwardly through a cloaca. At certain spots
the surface of the bone looks as if it were corroded and
in a necrotic state.

Tab. 44.

Tab. 45.

Explanation of Plate 45.

ARTHRITIS DEFORMANS.

The lower epiphysis of the femur shows a high degree of deforming bone proliferation, the result of an ossifying hyperplasia of the articular cartilages, in the course of which the proliferative process has spread to the neighboring periosteum. Those portions of the bone which are contiguous to the articular surfaces are also covered with irregularly shaped osteophytes—some with jagged outlines, others thorn-shaped. That portion of the bone which has been deprived of its covering of cartilage is now covered with the products of a sclerosing osteitis.

Explanation of Plate 46.

The body of the vertebra, and particularly the lower half, is in great measure destroyed. The portions of bone which were affected by the purulent and cheesy inflammation (tuberculous osteomyelitis) and the necrosis, but were not destroyed, are easily friable and present an uneven and spongy looking surface. The spinous process and one of the transverse processes also show traces of having been attacked by the disease and of having undergone some softening. The secondary angular bending of the lower part of the spinal column in a forward direction has already begun to show itself. (Preparation belongs to the Pathological Institute.)

Tab. 46.

Tab. 47.

Explanation of Plate 47.

TUBERCULOUS BONE ABSCESS IN THE LOWER PORTION OF THE HUMERUS. (From a Child.)

The tuberculous caries has, in this case, led to the formation of an abscess cavity, which is situated in the substance of the very much expanded and thickened lower portion of the humerus. This abscess has broken through to the surface, and, after partially destroying the epiphysis, has involved the elbow-joint. The thickening of the surrounding bone substance is due partly to hyperplastic osteitis and partly to periostitis.

Explanation of Plate 48.

MYELOGENOUS HEMORRHAGIC SARCOMA OF THE HUMERUS, WITH CENTRAL SOFTENING.

The upper third of the bone (right humerus) is the seat of a tumor larger than one's fist, of a dark brown color, and of rather soft consistence. When pressed upon it gives forth a crepitating, grating sort of a sound, which is due to the presence of some fragments and thin scales of bone in the shell-like walls of the tumor. In the centre of the growth, in the place of the bone which has become disintegrated, there is a cavity, larger than a goose's egg, which is filled with blood and blood-tinged remains of tissue. The head of the humerus is in a necrosed state in its interior, while its convex articular surface, to a depth of a few millimetres, is still intact and may easily be bent when pressed upon. That portion of the shaft of the bone (middle third of the humerus) which borders upon the tumor is fissured and somewhat disintegrated. The picture presented under the microscope is that of a very vascular myxo-sarcoma. Tumors of a similar nature were found on the left humerus, on the left femur, and on one rib.

The patient upon whom the above-described tumor was found was a man fifty-three years old (Journal, No. 383, 1897), who stated that he had been ill only two months. At first, he had suffered from severe pains in the upper epiphyses of the three tubular bones mentioned above. Then, in the course of a few days, tumor-like swellings had made their appearance. The skin, at the affected spots, was found to have a dark-red color, and distinct fluctuation was felt. When the parts were incised apparently nothing but blood escaped. Central hemorrhagic bone sarcomata, such as we are now considering, were formerly termed hæmatomata of bone (Volkmann) or bone-aneurisms.*

* A more detailed description of the present case will be found in the dissertation of Emil Walther: "Zwei Fälle von myelogenem Osteo-Sarcom." Munich, 1888.

Tab. 48.

Tab. 49.

a.

b.
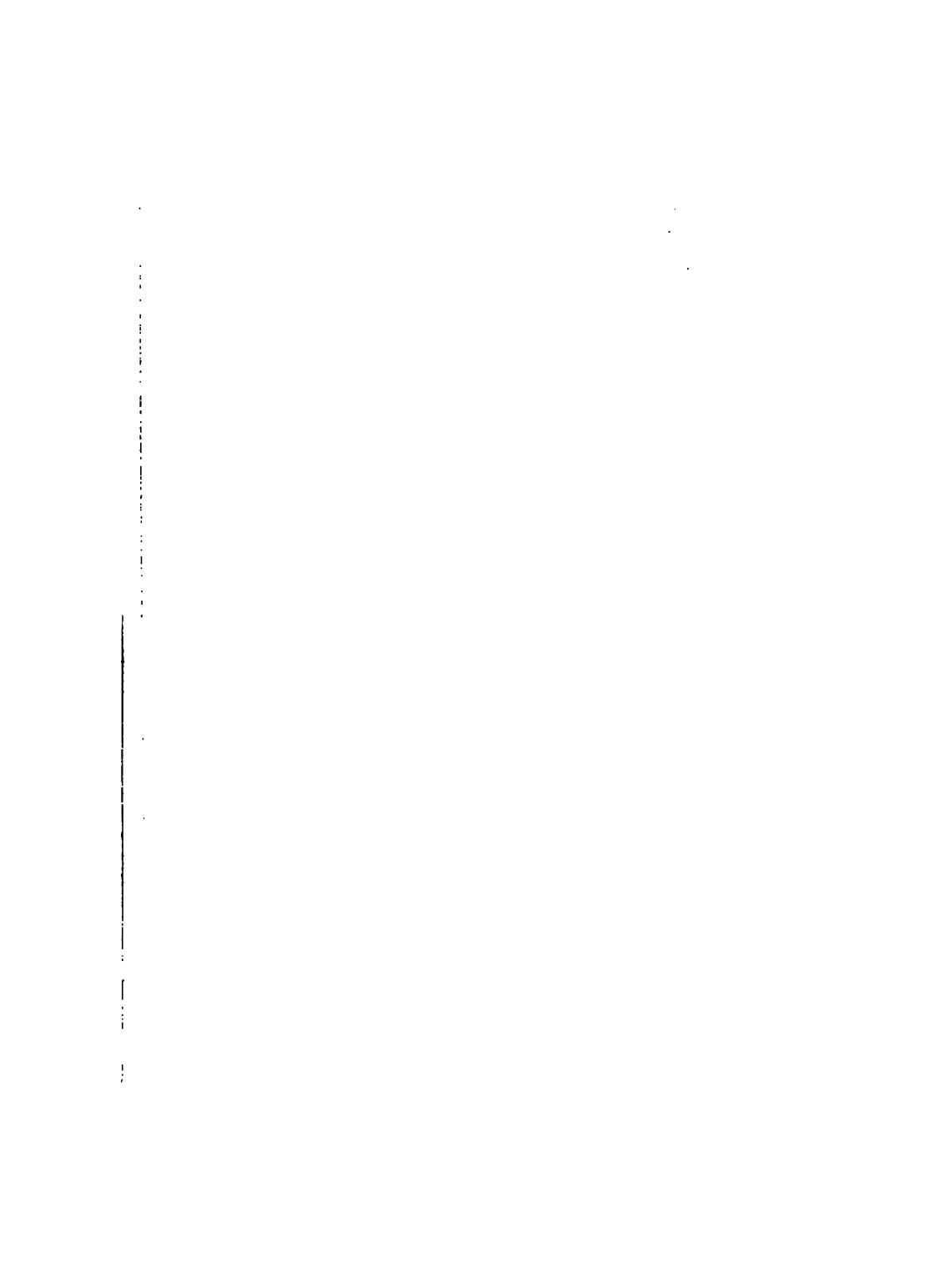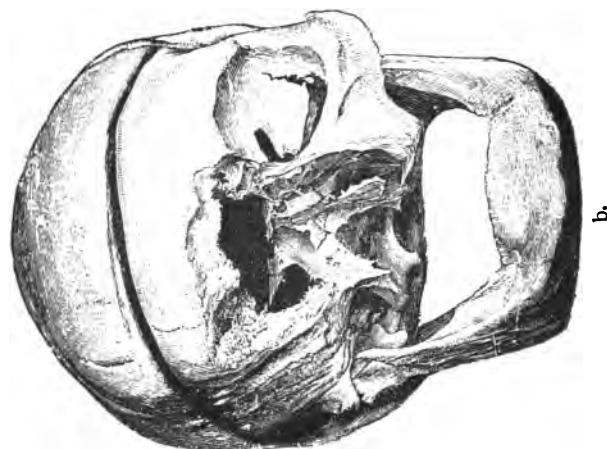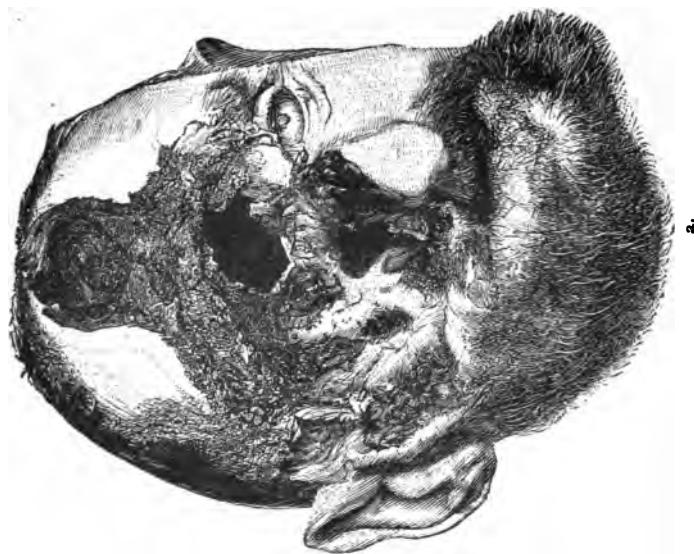
Explanation of Plate 49.

CARCINOMA OF THE SKIN OF THE FACE, WITH IN-
VOLVEMENT OF THE UNDERLYING BONY STRUC-
TURES. (Duration, about twenty-one years.)

The patient was a man, sixty-four years of age. When he
was twenty-six years old a wart, a little larger than the head of
a pin, appeared upon the lower border of the right lower eyelid.
Most of the time this small wart was in an inflamed condition,
and the skin, for a short distance around it, was somewhat red-
dened. A little moist secretion came from the growth. By the
time he reached his forty-third year the ulcer, which had been a
good deal irritated by the frequent removal of the crusts which
formed upon it, had already increased greatly in size, both
superficially and in depth. It had advanced in the direction of
the mouth. By the end of another year it had so increased in all
its dimensions that it could hold the last joint of a man's finger.
While at first the disease had advanced very slowly, it began at
this period to take on a more rapid rate of growth, so that at the
end of the next succeeding ten years the ulcer had attained greatly
increased dimensions. Seven years before his death the loss of
one eye took place; then, later, pieces of necrosed bone escaped
from the wound; and finally, toward the end of his life, the
brain itself, robbed of its protecting dura, projected as a pulsat-
ing tumor from the bottom of the ulcerated cavity. During the
last two years of life frequent and copious hemorrhages from the
ulcer took place.

The cancerous ulcer (Plate 49, Fig. *a*) involves the entire right
side of the face, and extends upward, upon the right half of the
forehead, in the form of a diverticulum as large as a silver dollar,
to and a little beyond the margin of the hairy scalp. At some
spots it has laid bare the surface of the bone. With the excep-
tion of its tip end the entire nose is destroyed. That part of the
face which is eaten away is about as large as a man's fist. This
excavation is limited, on the outer side, by the ascending ramus
of the lower jaw; below, by the hard palate; toward the left
side, by the septum narium; and above, by the frontal bone.
At its fundus the excavation extends as far as to the pterygo-
maxillary fossa. The walls of the cavity are covered with
masses of cancerous and necrotic tissues. Upon the macerated
skull (Plate 49, Fig. *b*) it can be seen that the entire superior
maxilla of the right side, with the exception of the hard palate,
has been destroyed; and, further, that the ascending portion of
the palate bone, the turbinated bones, the lacrymal bone, the
ethmoid bone, and both nasal bones have met with the same fate.
The left [right?] orbital cavity is entirely destroyed, and toward
the median line the roof of this cavity and the adjoining nasal

portion of the frontal bone are deficient, thus affording an open-
ing into the cranial cavity fully as large as a fifty-cent piece.

At the post-mortem examination no metastatic foci could be
discovered in any part of the body, and both the submaxillary
and the cervical lymph glands were found—both macroscopi-
cally and microscopically—to be normal.   Specimens taken
from both the base and the margins of the ulcer were submitted
to a microscopic examination, and its carcinomatous character
was thus established beyond all doubt.   (The characteristic
epithelial plugs and cylinders were found in these specimens.)
(A more detailed description of this case will be found in the
dissertation of Fritz Zorn, entitled : "Seltene Fälle von Gesichts-
krebs mit ungewöhnlich langer Dauer," Munich, 1891.)

Explanation of Plate 50.

SUBCORACOID DISLOCATION OF THE HUMERUS, OF
LONG STANDING; FORMATION OF A NEW SOCKET
ON THE SCAPULA AND WEARING AWAY OF THE
HEAD OF THE HUMERUS.   (After Helferich.)

Fig. *a* shows both bones—as seen from in front—in their posi-
tion of dislocation.   The head of the humerus covers the region
of the glenoid fossa and rests upon the anterior surface of the
neck of the scapula, beneath the coracoid process.   The free an-
terior surface of the head of the humerus, with its covering of
cartilage tissue, is visible, and so also is the border of the newly
formed mass of bone, on the neck of the scapula, which sur-
rounds the new joint socket.   The humerus occupies a position
of slight abduction.   The mobility of this abnormal arrange-
ment of the joint is extremely limited.

In Fig. *b* the two bones are represented in such a manner that
the scapula is seen from in front, just as it is in Fig. *a*; while
the humerus, having been rotated through an arc of 180°, pre-
sents its posterior surface—that which normally faces the scapula
—toward the eye of the observer.   In this view the glenoid fossa
is seen (on the scapula) from one side, and therefore a good deal
foreshortened.   Its anterior border is also seen to be considerably
worn away ; and immediately next to it, toward the median line,
comes the new joint socket, surrounded by its irregularly out-
lined wall of bone.   On the humerus may be seen an excavation
which owes its origin to the wearing away of the bone caused by
pressure upon the margin of the glenoid fossa ; and in the local-
ity which corresponds to the anatomical neck a few masses of
newly formed bone tissue, like those which are characteristic of
arthritis deformans, are visible.

Tab. 50

a

b

Tab. 51.
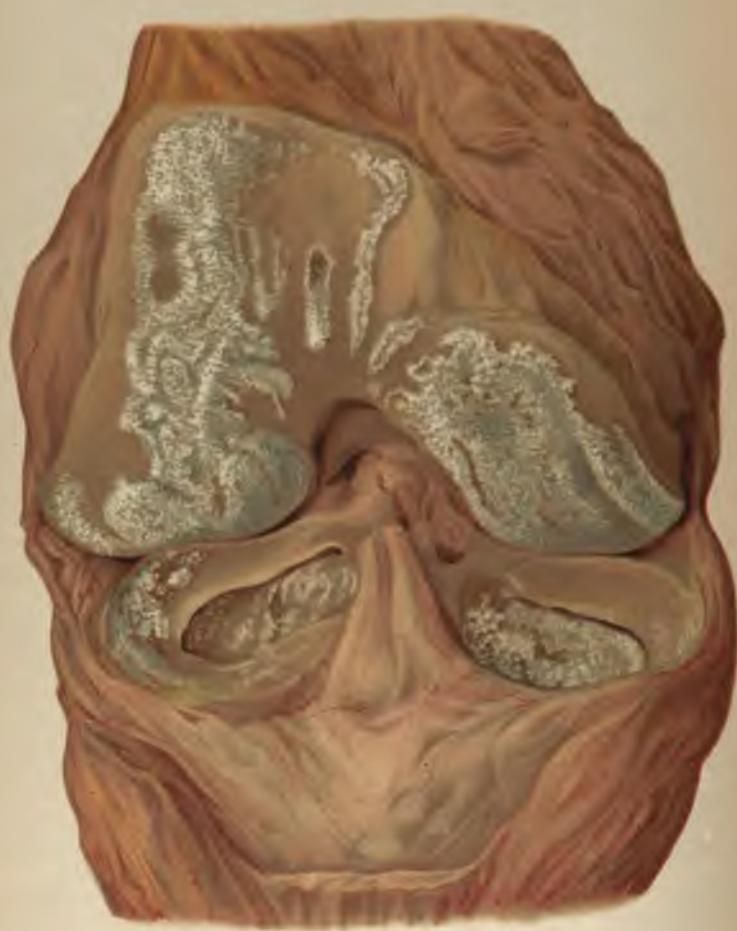
Explanation of Plate 51.

GOUTY INFLAMMATION OF THE KNEE-JOINT; GONARTHRITIS URICA.

In the synovial membrane of the knee-joint, which had been laid open, there were found whitish and bluish-white deposits of uric acid which looked like plaster of Paris. Similar deposits were also found in various other joints (polyarthritis urica).

The pathological changes which, in this patient (a baker, forty-five years of age), led to a fatal issue, were the following: Granular atrophy ("gouty kidney") of the right kidney, with congenital absence of the left kidney and corresponding ureter; lobular pneumonia on both sides, with commencing pleurisy; a high grade of general anæmia; and concentric hypertrophy of the left ventricle of the heart. The patient, who, toward the end of his life, had become a drinker, acquired the gout in 1872 (twenty-two years before his death), as the result—so he said—of the campaign of 1870-71. From that time onward he had had gouty attacks, almost every year, in the spring and autumn. In these attacks the joints of the hands and feet had always become swollen. No evidence of heredity could be discovered. (No. 289, 1894.)

Explanation of Plate 52.

TUBERCULOUS FUNGOUS INFLAMMATION OF THE KNEE-JOINT (GONARTHRITIS).

The knee-joint presents the condition of a fungous synovitis. The cavity of the joint contains a moderate quantity of thin pus, mixed with flakes of fibrin. The larger part of the synovial membrane is covered with grayish-red, succulent, sarcoma-like, spongy granulations, which in some places measure half a centimetre, or even more, in thickness. Scattered throughout these rather low, hummocky products of inflammation, which under the microscope present the picture of a granulation tissue rich in cells, there are tuberculous nodules, which appear, even to the unaided eye, as prominent, opaque-yellow, nodular growths. The cartilage tissue which covers the patella and the epiphysis of the femur remains almost entirely intact, owing to the fact that the process is of comparatively recent date.

Tab. 52.

Lith.Aust.v.F.Reichhold, München

PATHOLOGICAL ANATOMY.

Urinary Apparatus.

DISEASES OF THE KIDNEYS.

CIRCULATORY DISTURBANCES.

VENOUS HYPERÆMIA OF THE KIDNEYS.

(Passive Congestion of the Kidneys. Plate 1.)

THIS condition may develop in connection with any
one of a number of disturbances of the circulation,
such as diminution of the power of the heart, idio-
pathic hypertrophy of this organ, valvular defects,
and pulmonary affections (emphysema, for ex-
ample).

In *acute hyperæmia* the kidneys will be found to be
enlarged, highly congested and succulent, and col-
ored a dark bluish-red.

In *venous hyperæmia of long standing* the kidneys
are enlarged, and they present a dark bluish-red col-
oration, especially in the region of the medullary
substance. At the same time they will be found to
possess a firmer consistence than usual (cyanotic
induration). When the hyperæmia lasts for years
the cyanotic and indurated kidney (Plate 1) grad-
ually undergoes atrophy. Not only does the connec-

tive-tissue framework increase in bulk, and the veins and capillaries become dilated, but there will also be a certain amount of inflammatory action—a limited glomerulitis—terminating in atrophy. In the midst of the tissue, which has become cyanotic and indurated, there will be found, in the cortical region, scattered areas in which the tissues have undergone atrophy as the result of an antecedent inflammation; while at the same time the medullary substance, itself also in an indurated condition, presents evidences of a diffuse atrophy. The dark-colored and tough kidney, whose surface is studded with slightly elevated nodules, grows smaller and smaller. This is the *congested and contracted kidney* shown in Plate 1. Now and then it will be found that the cyanotic hue of the diseased kidney has become very much paler. This is especially true of individuals whose fundamental disease (some form of heart affection) has terminated in a general anæmia and cachexia of long duration.

We may accordingly speak of a granular atrophy of the kidney, which develops from a cyanotic induration, and is often mistaken for the true contracted kidney. The granules at first are of a darker coloring; at a later stage, however, they present a pale appearance, but yet they do not lose entirely their livid hue. The most important alterations in this variety of contracted and congested kidney are the following: Dilatation of the capillaries and veins; thickening of the walls of the capillaries; pressure atrophy of numerous uriniferous canaliculi; atrophy of glomeruli with thickening of their capsules; and thickening of the walls of the blood-vessels (the intima being involved only in the case of arteries, while

the adventitia may be thickened in both the arteries and the veins).

Among the functional disturbances which accompany this form of congested kidneys may be mentioned diminution of the quantity of urine excreted, increased specific gravity, formation of an abundant sediment, slight albuminuria, and the occasional presence of a few hyaline casts in the urine. In that form of nephritis which is due to venous congestion of the organ, epithelial casts and red blood corpuscles will also be found in large numbers in the urine.

Anæmia of the kidneys is found as an accompaniment of general anæmia. The pale gray color which characterizes this condition is found either to extend uniformly throughout the kidney or to be confined to the cortex. In certain parenchymatous inflammations which pursue a chronic course, there will generally be found, in addition to the pale aspect due to anæmia, a certain amount of fatty degeneration. (The so-called large white kidney is an instance of this.) In fatty and amyloid degeneration the kidney is indeed anæmic, but it is also at the same time enlarged and presents a more spotted appearance.

Hemorrhages in the Kidneys.—These are often to be demonstrated only by aid of the microscope. They are a common occurrence in acute inflammations of the kidneys (hemorrhagic nephritis) and in the general condition termed "hemorrhagic diathesis." If the blood which has escaped from the vessels is confined inside the Malpighian capsules, the latter will appear as small red dots scattered uniformly throughout the cortex. But when the blood escapes into the uriniferous canaliculi, the appearance presented will

be that of fine striæ and flecks. Traumatic hemor-
rhages occur in consequence of injuries to the kid-
neys, as after a fall from a height, gunshot wounds,
etc.; and in connection with these it is not unusual
to find that there has also been an escape of blood
underneath the capsule (perirenal hemorrhage) and
into the pelvis of the kidney (hæmaturia).

Embolism of branches of the renal artery leads to the
formation of hemorrhagic infarctions, which are char-
acterized by their wedge-like shape (the base or broad
part of the wedge being located at the surface of the
kidney, while the apex corresponds to the point where
the embolus plugs the blood-vessel). Embolic infarc-
tions (Plate 2) vary in size, but the majority of them
are no larger than a pea or a small bean. They are
of a pale color (anæmic infarction) with a red, hemor-
rhagic border zone (see Plate 2). The tissues which
are thus deprived of their arterial blood-supply rap-
idly undergo anæmic necrosis and fatty degeneration.
In the place of these tissues which have died and have
been gradually absorbed, there will be found a certain
amount of cicatricial fibrous tissue, the retraction of
which causes the formation of a depression or pit of
greater or less extent. Small embolic infarctions are
often composed wholly of escaped blood. Kidneys
containing cicatrices that owe their origin to repeated
embolisms are oftenest encountered in individuals
who have had valvular diseases of the left side of the
heart, or ulcerous sclerosis of the aorta, or the forma-
tion of thrombi in the left heart chamber or in the
aorta. When the embolus is of an infectious charac-
ter, as in septico-pyæmic processes, an abscess is
sure to develop at the seat of the infarction (Plate

3) ; and in this case the surface of the kidney is likely to present, after the capsule has been stripped off, a certain number of purulent foci, each of which has more or less the shape of a wedge and is surrounded with a red zone.

NEPHRITIS.
(Inflammation of the Kidneys.)

Two kinds of inflammation of the substance of the kidney are recognized, viz., the idiopathic and the secondary. In the former the disease is apt to run a chronic course, while in the latter the nephritis is generally of an acute nature. Then, besides, it is customary to distinguish a parenchymatous and an interstitial variety of inflammation of the kidney, not to mention a number of mixed and transitional forms of the disease. The more rapid the development of a nephritis, and the shorter the course which it runs, the more likely is it that we shall find the excreting parenchyma alone involved; while in a nephritis of long duration the degree in which the connective-tissue framework of the kidney participates in the inflammation will be directly proportionate to the duration of the disease.

Acute Parenchymatous Nephritis.
(Plate 7 and Plate 9, Fig. *a*.)

This disease may be caused by a number of infectious and toxic agencies, such as scarlet fever, various febrile processes (sepsis, typhoid fever, diphtheria), different poisons (*e.g.*, cantharides), etc. ;

and it may develop in the course of pregnancy, as
well as without any demonstrable cause (a chilling
of the body?).   (Plate 7.)

In the *first stage* of the condition commonly termed
cloudy swelling the kidney appears to be enlarged and
its capsule can easily be removed.   The color of the
organ is a dull grayish-red or a pale gray.   In a
section the cortical zone will be seen to be broader
than normal, and usually paler than the medullary
substance.   A decidedly œdematous appearance of
the cortex will also be observed, and the normal stri-
ations will be seen somewhat indistinctly.   By pass-
ing the edge of the scalpel over the cut surface of the
kidney, under moderate pressure, an abundance of an
opaque grayish fluid will be obtained.   Pressure upon
the papillæ affords escape to a grayish-white juice
which contains large quantities of cast-off epithelial
cells (indicative of a desquamative or catarrhal ne-
phritis).   In specially severe cases blood escapes
from the vessels by diapedesis into the capsular space
of the glomeruli and also to some extent into the uri-
niferous canaliculi (hemorrhagic nephritis).   The
epithelial cells are swollen as if in a dropsical con-
dition, and small particles like dust are scattered
throughout their protoplasmic contents.   Further-
more, many of them show evidences of disintegra-
tion.   Hyaline and epithelial casts are found in the
urine; the hyaline ones owing their origin to the
solidification of albumin, particularly in the loops
and intercalated portions.

Under favorable conditions, as when the primary
process shows a tendency to subside, all these path-
ological changes disappear in a short space of time,

and newly reproduced epithelium speedily takes the place of that which has been cast off.

Under unfavorable conditions the stage of cloudy swelling is succeeded earlier or later by that of fatty degeneration.

In the stage of *fatty degeneration* the enlarged kidney presents the following characteristics: Its color as a whole is pale gray; the tissues contain scarcely any blood, and they break down easily; and the organ appears decidedly spotted, by reason of the fact that there are small areas of a pale yellow or somewhat whitish color—spots which correspond to the regions where the fatty degeneration of the epithelium has advanced to a higher degree. (Plate 9, Fig. *a*.)

In quite a number of cases the process advances still further, and there is established either a *subacute* or, more rarely, a *chronic parenchymatous nephritis—* a form of renal disease which is often also termed the "large white kidney" or the "large spotted kidney." (Plate 8.)

In this last form of renal disease the kidneys are half as large again as they normally should be, and in a few instances they are even larger. Their color is a washed-out whitish gray, and they are distinctly spotted. The normal striations of the cortex are not distinguishable. In respect of their consistence these organs are generally a little tough. Under the microscope the following alterations may generally be recognized: Swelling, cloudiness, and proliferation of the epithelial cells of the uriniferous canaliculi; fatty degeneration of these cells, distributed unsymmetrically throughout the tissues; frequently proliferating

glomerulitis, associated with decided enlargement of
the glomeruli, which are very rich in nuclei through
the proliferation of the endothelium. The inter-
stitial tissue is often markedly infiltrated with cellu-
lar elements, as a result of which condition the struc-
tural details do not stand out distinctly; the tissues
look more like those of a sarcoma. When these alter-
ations are found, it may be assumed that a diffuse
interstitial inflammation has been engrafted upon the
parenchymatous variety.

Chronic Interstitial Nephritis.

Indurative nephritis; granular contracted kidney;
true contracted kidney; Bright's disease; atrophic
renal cirrhosis.   (Plate 4.)

This disease is characterized by an insidious begin-
ning and a chronic course. The anatomical changes
which accompany it are the following: The kidneys
are markedly reduced in size, sometimes—in far-ad-
vanced cases—even to one-third the normal size. The
capsule is thickened and hard to separate from the
kidney, small portions of the cortex often being found
adhering to its inner surface after it has been removed.
The organ itself presents a finely granular surface, of
a waxy or whitish-gray color; the projecting gran-
ules generally displaying an appreciably brighter
shade of color than the surrounding tissue, which is
somewhat cicatricial in character and consequently
occupies a lower level. The kidney is so tough and
hard that it cuts like a piece of India rubber. The
surface of the section has a pale gray color, and nei-
ther blood nor juices of any kind escape from it under
pressure. The cortical zone is much narrowed, to

one-half and sometimes even to one-third the normal breadth, and the line of demarcation between it and the pyramidal substance is almost obliterated. Under the microscope it will be seen that the stroma has undergone a marked increase, and that it is infiltrated with an abundance of round cells, which are aggregated in more or less well-defined areas. The Malpighian bodies will be found in general to possess thickened capsules, and they themselves present every possible degree of contraction and hyaline atrophy. In consequence of the condensation of the parenchyma of the cortex the Malpighian coils are brought so much nearer together that, instead of two or three, sometimes as many as six or even eight may be seen at the same time in the field of vision. The walls of the arteries are thickened, particularly the adventitia and the intima. Small cysts containing serum are often seen scattered throughout the atrophic tissue. In the earlier stages of the process, which now and then come under our observation, as when a patient dies of some intercurrent disease like tuberculosis, pleurisy, or apoplexy, the picture presented is that of an interstitial nephritis, occurring perhaps more often in the form of separate centres of inflammation, and characterized by the presence of very small granulations upon the surface of the kidney (when freed from its capsule) and by only a slight diminution in size of the organ.

The question of the *etiology of the genuine contracted kidney* is surrounded by a great deal of obscurity. The disease occurs about twice as often in men as in women. The period of life at which it develops most commonly is that from the fortieth to the sixtieth

year (about fifty per cent of all cases), while more
rarely the disease is observed between the ages of
thirty and forty (from fifteen to twenty per cent of
all cases). Aside from individual predisposition, the
most effective among the etiological factors are un-
doubtedly certain toxic influences, chief among which
may be mentioned *chronic lead poisoning* (house paint-
ers, pottery glazers, type setters); excessive use of
alcohol, especially when associated with the habitual
indulgence in strongly spiced and irritating articles
of food which contain a large percentage of albumin
(luxurious living); and, finally, a uric-acid diathesis
("gouty contracted kidney"), in which condition the
uric acid acts like a poison upon the kidney tissues.
In *arthritic nephritis*—both with and without any in-
volvement of the joints—there will often be found in
the urinary canaliculi of the medullary substance,
which looks as if it had been sprinkled with small
white dots, deposits of the urate of sodium in the form
of rhombic columns and crystals. The disease is also
favored by certain vascular conditions, such as pre-
senile arterio-sclerosis, and diffuse hypertrophy of
the heart, involving both ventricles and associated
with a chronic increase in pressure in the arterial
system. As a secondary phenomenon, which occurs
with almost perfect regularity, should be mentioned
decided hypertrophy and moderate dilatation of the
left ventricle; and in many cases this progressive and
incurable pathological process is interrupted by some
fatal intercurrent disease, like apoplexy, tuberculosis,
pleurisy, pneumonia, etc. As concomitant symptoms
of this pernicious disease of the kidneys may be men-
tioned progressive emaciation, polyuria, moderate al-

buminuria, disturbances of vision, and uræmic mani-
festations.

The *contracted kidney of secondary development* (in
contradistinction to the "genuine contracted kidney")
is encountered much more rarely. It develops from
forms of kidney inflammation which at first run an
acute, subacute, or subchronic course, and involve
mainly the parenchyma. Thus, for example, a ne-
crotic process involving the epithelial structures may
lead to secondary interstitial nephritis atrophicans.
A smooth or only slightly granular form of contracted
kidney sometimes develops—although rarely, it must
be confessed—from the large white kidney. The
pathological process which takes place in these cases,
and which extends uniformly throughout the paren-
chyma of the kidney, gives rise to a correspondingly
uniform and widespread atrophy, or at the most to a
slightly granular surface of the organ. In this same
category also belongs the slightly granular contracted
kidney which is associated with a condition of chronic
congestion—a form of pathological kidney which is
often mistaken for the genuine contracted kidney.

The *arterio-sclerotic contracted kidney*, which is ob-
served in persons of an advanced age (beyond the
sixtieth year), and frequently even in younger in-
dividuals who are affected with a syphilitic endar-
teritis, also presents certain points of resemblance to
the genuine contracted kidney. It is in this variety
that the very first alterations are to be looked for in
the walls of the smallest arterioles (obliterating arte-
ritis). Furthermore, on the surface of the kidney
shallow, irregularly shaped depressions (due to cica-
tricial contraction) may often be seen; and in the

parenchyma of the organ evidences of inflammation
of the connective-tissue framework will be observed
in close proximity to simple atrophy and destruction
(through wasting away) of numerous glomeruli—
alterations which warrant the belief that arterio-
sclerotic changes and a proliferative inflammation
which terminates in atrophy often advance hand-in-
hand as parallel processes.

TUBERCULOSIS OF THE KIDNEYS.
(Plate 12.)

Tuberculosis of the kidneys is very rarely encoun-
tered as a primary lesion. It is usually a secondary
phenomenon, which owes its origin to the presence of
tubercle bacilli in the blood, and which also may be
looked upon as one of several evidences of a general-
ized miliary tuberculosis. Or else it may be consid-
ered in the light of a terminal and accidental mani-
festation of this disease in the course of a chronic
pulmonary tuberculosis, the tubercles occurring in
the kidney in the form of disseminated miliary foci,
which occupy every section of the organ, but are
especially numerous in the cortex and just beneath
the capsule.

The tubercles themselves vary greatly in number
and in size, some being no larger than a milletseed
and presenting a grayish exterior with a whitish or
yellowish-white centre, while less frequently they are
encountered in the form of subacute granulomata,
varying in size from that of a hempseed to that of a pea.

The *chronic inflammatory form of renal tuberculosis*
is encountered as one of several manifestations which
characterize a tuberculosis of the urinary apparatus

—a disease which as a rule manifests this peculiarity, viz., that the tubercles first invade the prostate and then the remaining parts in the following order: testicles, urinary bladder, ureters, pelvis of the kidney, and finally the parenchyma itself. It is only in rare cases that the kidney is the organ first affected. When this happens the process begins at the base of the pyramids, extends to the papillæ, and then in turn involves—in a downward direction—the pelvis, the ureters, the urinary bladder, and the urethra. In such a case it will be found that there are fewer tubercles in the bladder than in the kidney—a circumstance which, in any case of genito-urinary tuberculosis, may be taken as proof of a downward course in the advance of the disease. In renal phthisis the kidneys are generally enlarged, and the capsules adherent. When the organ is laid open by a cut with the knife, it will be found that only a narrow remnant of kidney substance still remains underneath the capsule. The pelvis, which is somewhat distended, contains purulent and cheesy products of a retrograde metamorphosis. In fact, it is a regular cavern, whose walls are composed of friable, yellowish-white, cheesy masses, scarcely any traces remaining of the papillæ and medullary substance. The ureters are converted into tough and rigid cords, with much narrowed lumina; and the mucous membrane lining the latter is in some places merely thickened, while in others it is converted into fissured, friable masses of necrotic and cheesy-degenerated tissue. Besides pus and cheesy products tubercle bacilli are apt to be found in the urine, but they are sometimes entirely lacking. (Plate 12.)

Healing of tuberculous disease of the kidneys, with the eventual formation of cicatricial tissue, cysts, or chalky masses, is a termination which can scarcely be expected.

These destructive processes in the kidneys are very often accompanied by simultaneous disease of the genital organs—*genito-urinary tuberculosis.*

DEGENERATIVE AND RETROGRESSIVE PROCESSES IN THE KIDNEYS.

Among the changes of this nature in the kidneys the commonest is *fatty degeneration* ("fatty kidney"), which may either develop rapidly and run a brief course (as, for example, in some cases of poisoning [phosphorus] and in certain infectious diseases), or may develop somewhat slowly as the final stage, or as an accompanying phenomenon, in a variety of inflammatory processes, such as subacute and subchronic parenchymatous nephritis. It is also observed as an accompaniment of the amyloid kidney. (Plate 9, Fig. *a.*)

The fatty change takes place first in the epithelial cells of the urinary canaliculi, and it either affects them symmetrically and diffusely throughout the entire organ (as in the case of the fatty kidney caused by phosphorus poisoning), or else it is confined to certain spots or larger areas. When the organ is diffusely affected the enlarged kidney presents a uniformly pale or a somewhat whitish-yellow color; it also contains very little blood, but rather an abundance of juices. Finally, the region of the cortex is much broader than normal, and the striations are

somewhat washed out and indistinct. If the edge of the knife is passed lightly over the cut surface of the kidney a cloudy, rather thickish juice will be obtained. When the fatty degeneration occurs in scattered areas, the organ presents a more spotted appearance, and small, ill-defined yellow and yellowish-white spots will be found scattered throughout the pale, swollen, and juicy kidney substance.

Amyloid Kidney.

In that form of degeneration which produces the amyloid or the lardaceous kidney, the organ is enlarged, of a dull gray and grayish-yellow color, and marked with numerous fine spots which represent disseminated areas of a secondary fatty degeneration. So far as the size and the color are concerned this variety of kidney shows a certain resemblance to the large white kidney (subchronic parenchymatous and interstitial inflammation). The amyloid degeneration generally begins in the capillary coils of the glomeruli and in the finer arteries; but, when the disease has reached an advanced stage of its course, the amyloid change involves not only the interstitial tissue but also the tunica propria of the urinary canaliculi. The lardaceous metamorphosis develops in the course of serious chronic disturbances of nutrition; as, for example, in chronic pulmonary tuberculosis, in tuberculous bone disease, in old syphilitic cases, and in chronic suppurations, especially of bony structures. The occurrence of the disease without any known cause is a rare event. The possibility of a cure is scarcely to be thought of.

ABNORMAL DEPOSITS.

Uric-acid infarctions in the form of yellow, brick-red, or yellowish-red bands in the papillæ and pyramids are seen oftenest in newborn children who have been alive at birth (and only very exceptionally in children born dead). The same remarks are true of those cylindrical or at times gland-shaped masses, composed of the urate of ammonium, which are seen in the urinary canaliculi (Plate 9, Fig. *b*). In doubtful medico-legal cases the presence of a uric-acid infarction—indicating, as it does, an abundant production of uric acid in the first few days of life—may be considered as evidence in favor of the view that the infant was born alive. In the gouty kidney (nephritis arthritica) deposits of sodium urate are also sometimes found, especially in the urinary canaliculi of the medullary substance.

Calcareous deposits are now and then observed in the medullary substance (at the points of the papillæ) as a phenomenon belonging to no particular disease, and also sometimes in the loop-shaped canaliculi in chronic nephritis. In this condition we are dealing either with a calcification of the tunica propria which has lost its epithelium, or with a deposit of the chalky material either in the calibre of the urinary canaliculi or inside the capsules of the glomeruli. Then, besides, acute chalky infiltrations of the kidney are sometimes observed in cases of corrosive-sublimate poisoning. (Plate 7.)

Hæmoglobin infarctions, which are observed in the form of brownish or yellowish-brown masses of hæmo-

globin, deposited in the urinary canaliculi of both the cortical and the medullary portions of the kidney, occur in certain forms of poisoning (chloride of calcium, arseniuretted hydrogen; poisoning by one of the poisonous varieties of the Morchella fungus; transfusion of another person's blood, etc.), and also in cases of severe burns. The associated hæmoglobinuria is accordingly a symptom of a great variety of morbid processes. It also sometimes occurs in the form of a periodic or paroxysmal hæmoglobinuria, as a consequence of exposure to cold or of excessive muscular exertion.

NEW GROWTHS OF THE KIDNEY.

They are not of rare occurrence. *Cysts* filled with serum or containing colloid, and representing retention cysts, are often observed at post-mortem examinations. They rarely occur singly, and they vary in size from that of a pin's head to that of a pea, or even some larger object. They generally occur in the course of a chronic interstitial inflammation, and in senile and arterio-sclerotic atrophy of the organ (acquired cystic kidney). As regards their mode of development, it may be said that they result from the obliteration of uriniferous canaliculi or from Malpighian bodies. Then, besides, one sometimes finds in a perfectly normal kidney a single large cyst—or even several of them—filled with a clear serous fluid and provided with a thin translucent capsule. (Plate 10, Fig *a*.)

In some instances the cystic development predominates to such a degree that only scanty remnants of kidney substance are left. When this condition of

affairs exists, it is proper to designate it by the term
cystoid degeneration of the kidney. Such cystic kid-
neys are observed in the newborn, and they may be
acquired at some later period of life. When they are
of congenital origin, these kidneys may be of such a
large size as to constitute in a pregnant woman a se-
rious obstacle to the birth of the child. A fœtal in-
flammation and obliteration of the papillæ (Virchow's
fœtal papillitis) is believed by some to be the cause
of this cystoid degeneration. It is also possible that
the condition may be really one of embryonal cyst-
adenomata.

Among the *non-malignant tumors* which are encoun-
tered in the kidneys there are, aside from the rarely
occurring fibromata and lipomata, certain growths
which develop from misplaced germinal portions of
the suprarenal capsules (struma renis aberrata, struma
suprarenalis). These tumors are situated in the cor-
tical substance, and project like hemispheres above
the surface of the organ. They have a whitish-yellow
color and a soft, marrow-like consistence; their boun-
daries are sharply defined, and they evidently grow
very slowly. When examined under the microscope
these tumors are seen to consist of fat-containing cells
arranged like a network, the structure resembling
closely that of the cortex of the suprarenal capsule;
or, in other words, that of alveolar adenomata which
are in part provided with papillæ. The rhabdomy-
oma, which rarely comes under observation, is also
of embryonic origin.

Of the *malignant tumors* the *sarcomas* are seen
oftenest. They occur in all their different varieties:
round-celled, spindle-celled, and large-celled sarco-

mas, angio-sarcomata and endotheliomata developing
from some point in the wall of a blood or a lymph
vessel, and those sarcomas which are often seen in
children in their first years of life (Plate 11). *Car-
cinoma of the kidney* is a much rarer tumor. It varies
in size from that of a fist to that of a man's head.
The surface of a section presents a whitish or a red-
dish color, and often shows the tissues to be inter-
spersed with cavernous spaces and accumulations of
blood (fungus hæmatodes).

In addition to these regular types, now and then
mixed forms of tumors are found in the kidney.

Multiple carcinomatous and sarcomatous nodules,
representing secondary and *metastatic* forms of tu-
mors, are often found in the kidney.

ABNORMAL POSITION OF THE KIDNEYS.

This pathological condition is either of congenital
origin or the result of forces operating later in life.
Generally only one kidney is affected, and in this
case it will be found lying at the upper margin of the
pelvis, on the promontory. The point of origin of
the renal vessels will also be found to be correspond-
ingly altered. When the two kidneys are fused into
one (the malformation known as the horse-shoe kid-
ney, or cake-shaped kidney), the organ may be found
lying in Douglas' sac or space, and in women, when
this is the case, its presence in this situation may
prove an obstacle to the birth of a child.

When the kidney is abnormally mobile, it is cus-
tomary to apply to it the term *wandering kidney* (ren
mobilis). The right kidney is the one usually affected

in this manner.   This mobility is to be attributed to
an abnormally large and relaxed capsule, and at the
same time the blood-vessels which furnish a certain
stability to the kidney will be found to have under-
gone more or less stretching and elongation.   In its
posterior portion the diaphragm protrudes forward,
almost in the form of a sac, into the peritoneal cavity.
The dislocated kidney lies somewhere between the
second and the fifth lumbar vertebræ.   When, as is
commonly the case, the right kidney is the one dislo-
cated downward and forward, one can feel it just below
the lower border of the liver.   Among the sequelæ of
this pathological condition may be mentioned the fol-
lowing: stretching and bending of the ureters and the
renal nerves, and pressure upon the intestines at dif-
ferent points.   The wandering kidney, which in its
slighter degrees is of common occurrence and scarcely
gives rise to any appreciable disturbances, is encoun-
tered at post-mortem examinations with surprising
infrequency.

In addition to congenital predisposition the follow-
ing may be mentioned as the chief causes of a mis-
placed kidney, a condition which is often associated
with enteroptosis and which is observed mainly in
women, and more especially in those who have
reached the middle and later years of life: Relaxa-
tion of the abdominal parietes, pressure exerted by
the adjacent liver, badly fitting clothing (for exam-
ple, a too tightly laced corset), repeated pregnancies,
and disappearance of the subperitoneal fat (as after
certain severe illnesses).

NEPHRO-LITHIASIS.

(Plate 10, Fig. *b*.)

Concretions and calculi are often found in the pelvis of the human kidney in middle life and in old age, and rather more often in men than in women. The concretions occur in the form of gravel-like masses, which are commonly composed of uric acid and its salts. In about twenty per cent of all cases the calculi and concretions are found in both pelves. Calculi are found in the actual substance of the kidney in about half of one per cent of all the dead bodies of adults.

The following varieties of calculi may be distinguished:

1. *Uratic calculi*, composed of uric acid and the urates. *Calculi of pure uric acid* are sometimes encountered. They are of moderate size and somewhat round, when they occur singly; or they are provided with facets when several of them are found together. Often they are branched, like a piece of coral, representing true casts of the pelvis of the kidney. They are of a yellowish-red or brownish color, and are composed of concentrically placed laminæ. In another group belong the calculi which are composed of the uric-acid salts (urates of ammonium and magnesium).

2. *Calculi composed of the oxalate of calcium.* These calculi are decidedly hard, and their surface is covered with small, rounded projections or regularly arranged prickle-like processes, of a blackish-

gray or a dark brownish-yellow color—the so-called
"mulberry calculi."

3. *Calculi composed of the phosphates and carbonates.*
They are of a whitish color, resemble chalk, and are
easily friable.   They usually develop in the course of
inflammations of the pelvis of the kidney, when the
urine undergoes certain chemical changes and alka-
line fermentation takes place.   Through the decom-
position of the urea carbonate of ammonium is formed,
and uric acid combines with ammonium to form the
urate of ammonium.   Then, again, by a combination
of the latter with the phosphate of magnesium the
triple phosphate is formed.

4. *Calculi composed of cystin* are soft and of the
color of yellow wax.   When broken they show a
crystalline fracture.

It is a common event to find *calculi composed of
laminæ of different materials.*   Thus, for example, the
inner layers may be composed of uric acid and the
urates, while the outer ones are formed of the oxa-
lates and triple phosphates.

The calculi which are formed in the pelvis of the
kidney may often, as is the case with gall stones,
give rise to no symptoms whatever; or they may, ac-
cording to their size and the character of their sur-
face, produce a variety of results.   Thus, for exam-
ple, they usually lead to dilatation of the pelvis of
the kidney, and when they are of large size they are
apt to produce atrophy of the organ itself.   Hæma-
turia and pyuria (pyonephrosis calculosa) may be
expected when the calculi irritate the surface of the
pelvic mucous membrane.   Then, again, a purulent
pyelitis may—following an ascending course—lead to

a papillitis and even to a nephritis, and these in turn
may induce a septicæmia or a uræmia; the latter con-
dition owing its origin chiefly to the fact that the
calculi, especially when they are small (from the size
of a pea to that of a bean), travel downward from the
pelvis and get tightly wedged in the channel of the
ureter. Finally, the renal calculi are usually the first
beginnings of what are afterward to be stones in the
urinary bladder. The development of the latter goes
on hand-in-hand with the deposit of organic material,
which furnishes a framework around which the stone-
forming materials may be deposited.

As one of the direct causes of the formation of a
calculus may be set down that anomaly of metabolism,
uric-acid diathesis, which is to be ascribed partly to
an inherited predisposition, and partly to external
influences (sedentary habits of life, too large a pro-
portion of albumin in the diet, excessive indulgence
in rich food).

DISEASES OF THE PELVIS OF THE KID-
NEY AND OF THE URETERS.

Dilatation of the pelvis of the kidney—hydronephrosis
—is a condition which is often found associated with
some obstacle which interferes with the evacuation of
the urine; as, for example, narrowing and closure of
the ureters under the influence of outside pressure,
and blocking of these channels by calculi which have
wandered from the pelvis of the kidney. According
to the situation of the obstacle which interferes with
the evacuation of the urine, the dilatation may in-
volve either one or both ureters. When both are

dilated, the cause is likely to be a carcinoma of the uterus (cervix) with implication of the wall of the bladder, or a stricture of the urethra, or a tumor of the prostate gland. As a result of the interference with the escape of the urine, the pelvis and calyces of the kidney become dilated and are eventually converted into a single sac-like cavity; and at the same time the papillæ are rendered broader and more flattened. The parenchyma of the kidney undergoes atrophy, and becomes tougher in consequence. Ultimately, a fluctuating sac, about the size of a man's fist, takes the place of the kidney, and in its wall some scanty remains of the organ may yet be found. The ureter is dilated to such a degree that it appears like a slightly tortuous canal of about the diameter of one's thumb.

INFLAMMATION OF THE PELVIS OF THE KIDNEY; PYELITIS AND PYELO-NEPHRITIS.

The mucous membrane of the pelvis of the kidney is swollen and unevenly reddened, and frequently shows small, disseminated, hemorrhagic foci. Generally the mucous membrane of the ureters presents very much the same pathological alterations. The material which coats the mucous membrane is a slimy, oftentimes purulent fluid. Different stages may be distinguished in the process, according to the nature of the cause from which it has originated. Thus, for example, in the beginning and in the milder degrees of the disease (as in renal calculi), the type is rather one of a desquamative and catarrhal character, while in the more severe forms the process pre-

sents the characteristics of a destructive and purulent inflammation. In those cases in which the inflammation is somewhat intense and lasts for a sufficient length of time, the process involves the parenchyma of the kidney—*pyelo-nephritis.*

The purulent pyelo-nephritis in a case like this is an *ascending process,* inasmuch as the inflammation can easily be traced from the bladder upward through the ureters to the pelvis. In the great majority of cases an analogous cystitis—one which agrees, both etiologically and as regards the pathological alterations, with that referred to above—furnishes the starting-point of the kidney disease. As illustrations may be mentioned the secondary cystitis which develops as a result of enlargement of the prostate, or that which owes its origin to a stricture of the urethra or to a paralysis of the urinary bladder; and, finally, the cystitis which develops when suppurative processes in the pelvic cavity lead to perforation into the bladder.

In *ascending pyelo-nephritis* the following pathological conditions are found: The pelvis of the kidney is filled with a discolored purulent fluid, the papillæ are covered with a coating of cast-off dead tissue, and, finally, throughout the medullary substance are scattered elongated abscesses, which give a striated appearance to the region. These abscesses, which are in the urinary canaliculi, contain large numbers of leucocytes and proliferating epithelial cells, and the tissues surrounding them are infiltrated with small cells. The pathogenic micro-organisms (usually bacterium coli and proteus) are collected together inside the urinary canaliculi and the lymph vessels, *but not*

in the blood-vessels. If, besides these, other micro-organisms (staphylococci, for example) are found, then the inference may as a rule be drawn that the case is one in which a mixed pyæmic infection has taken place. If the purulent foci extend from the papillæ to the surface of the kidney, the enveloping capsule may become involved in the inflammation; in other words, there may be a *purulent perinephritis* and *paranephritis.*

In *descending hæmatogenous metastatic nephritis* and *nephro-pyelitis* multiple abscesses will be found in the cortex directly beneath the capsule. These abscesses, which are either of embolic or simply of metastatic origin, belong among the manifestations of an infection of the blood—embolic septico-pyæmia. In this form of disease the pathogenic micro-organisms (pus cocci, pneumococci, typhoid bacilli) are found in the pus, in the inflamed tissues, and in the blood-vessels, secondary aggregations of the micro-parasites being only rarely found in the urinary canaliculi.

In both forms of the disease (ascending and descending pyelo-nephritis) the issue is almost always a fatal one, through sepsis, septico-pyæmia, and uræmia.

DISEASES OF THE URINARY BLADDER.

RUPTURE AND PERFORATION OF THE URINARY BLADDER.

Traumatic ruptures of the bladder—due to a blow, to a fall from a height, or to the fact of a person's being run over—occur when the organ is, at the time, more or less full. The rupture, which generally takes

place at the upper and posterior portion of the bladder, is either complete (intraperitoneal) or incomplete (subperitoneal or extraperitoneal). The tear begins within and extends outward. When the rupture is incomplete the urine is extravasated into the cellular tissue lying in front of the bladder and between it and the peritoneum. The consequence is a septic peritonitis. When the rupture is complete, blood and the contents of the bladder escape into the abdominal cavity, and the patient exhibits the pallor of anæmia.

Mechanical injuries of the bladder also occur not infrequently during labor and as a result of the various operations employed to aid in the delivery of the child. The ultimate results of these injuries are necrosis of the anterior or posterior wall of the bladder, vesico-vaginal fistulæ, and often pericystitis.

Hemorrhages into the mucous membrane of the bladder are observed in the course of inflammations of this organ, in individuals affected with the hemorrhagic diathesis, in cases of systemic poisoning, and in persons who are affected with villous tumors of the bladder or with enlarged veins about the neck of the organ (hemorrhoids of the bladder).

INFLAMMATION OF THE BLADDER; CYSTITIS.

There are both an acute and a chronic form of this disease, and the different degrees and modifications which are observed are numerous.

In *catarrhal cystitis* the mucous membrane of the bladder is red and swollen, and in its cavity will be found an increased quantity of mucus and cast-off

epithelium.   After the disease has gone on for a considerable length of time, difficulty will be experienced by the patient in voiding urine (retentio urinæ), and as a later result both the mucous membrane and the muscular coat of the organ will undergo hypertrophy (chronic hypertrophy of the walls of the bladder).

In the *more severe forms of cystitis* the exudation consists of pus alone.   Here and there a spot may also be seen where a layer of fibrinous material adheres to the mucous membrane or where a partial destruction of the latter has taken place (Plate 13). When the necrosis penetrates more deeply, an actual perforation of the bladder may be established.   The cast-off tissues, when the urine has undergone ammoniacal fermentation, present a grayish-white color, are filled with scattered concretions, and feel gritty when pressed between the fingers.   In such forms of infectious and diphtheroid cystitis—forms in which the pathogenic micro-organisms *bacterium coli* and *proteus* play a part—the urine generally shows an acid reaction, and the inflammatory changes in the mucous membrane must be looked upon as the direct product of the action of bacteria.   Processes which terminate in necrosis of the mucous membrane of the bladder are apt to develop when the urine undergoes ammoniacal decomposition, and the latter sets in when either a mixed infection of the urine takes place—as when proteus, diplococcus ureæ, staphylococci, and the saprophytic micro-organisms are associated with bacterium coli—or when a pure infection of this fluid with the diplococcus ureæ is effected.

Stagnation of the urine in the bladder plays an important part in the causation of cystitis.   The bac-

teria which gain an entrance into the bladder usually come from the urethra, the introduction of a catheter serving to convey them from the latter locality. But they are competent to set up a catarrhal inflammation of the bladder only when there exists at the same time stagnation of the urine (such as may result from a stricture of the urethra, from enlargement of the prostate, or from paralysis of the muscular walls of the bladder), or when the mucous membrane has been more or less injured (as may happen when a calculus is present).

There is such a disease as *gonorrhœal cystitis*, but it is of rare occurrence. The gonococci are then sometimes found, not only in the cavity of the bladder, but also in the capillaries and precapillary veins of the organ.

In *chronic cystitis* both the mucous membrane proper and the submucosa are markedly thickened, and so too is the muscular coat (trabecular hypertrophy of the bladder, the lattice-work bladder). In addition to the fact that it has an abnormal slatey-gray color, the mucous membrane presents the appearance of a grating or a honeycomb; this arrangement being due to the fact that the underlying bands of muscular fibres, which have become hypertrophied in an irregular manner, stand out prominently like the bars of a grating, and so communicate the same conformation to the mucous membrane which covers them. In the higher degrees of this hypertrophy the mucous membrane of the bladder presents an appearance very similar to that of the interior of the ventricles of the heart. Between the powerfully developed muscle bundles, pockets—some quite shallow and

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others of greater depth—are formed in the wall of the bladder. These, which are technically known as *diverticula*, vary in extent (see Plate 13). Now and then a case is observed in which a purulent chronic cystitis (both with and without diverticula) is associated with a purulent inflammation of the connective tissue surrounding the bladder—a para-cystitis which terminates in the formation of a distinct abscess—or with an inflammation of its peritoneal coat—a pericystitis.

Tuberculous cystitis is a disease of rare occurrence. The lesions usually found are small and medium-sized lentil-shaped ulcers with shallow cheesy floors and sharp indented borders. These ulcers rapidly increase in size through the confluence of two or more smaller ones. Catarrh of the bladder is usually present at the same time. Not infrequently tuberculosis of the kidneys, the prostate gland, the testicle, and the spermatic cord (genito-urinary tuberculosis) is associated with the bladder lesions.

URINARY FISTULÆ.

Among the different fistulous communications which are observed between the ureter, the bladder, and the urethra and neighboring organs, the *vesico-vaginal fistula* is the one most often encountered. These fistulæ often develop in a gradual manner; as, for example, when a carcinoma of the uterus invades the anterior wall of the vagina, and then, extending from here to the posterior wall of the bladder, gradually causes the normal tissues involved to break down. And then if, at the same time, the posterior wall of

the vagina and the anterior wall of the rectum become involved in the cancerous disease, a *vesico-vagino-rectal fistula* will be established.

Furthermore, vesico-vaginal fistulæ may develop as a result of an unnatural labor (through pressure of the child's head, causing necrosis of the soft tissues, in cases of contracted pelvis or of oblique face presentations), and especially after the forceps or the perforatorium has been used in an unskilful manner. They may also develop as a result of certain gynæcological operations. Involuntary evacuation of the bladder by way of the vagina, and incrustation of the latter with sedimentary deposits from the urine, are the usual results of this pathological condition. It also sometimes happens that an ascending purulent pyelonephritis develops in connection with the cystitis which is frequently associated with such a fistula.

NEW GROWTHS OF THE BLADDER.

Among the *benign tumors* that which is oftenest encountered in the bladder is the *villous papilloma* (papillary fibroma or fibro-epithelioma). These are delicate, spongy, pedunculate tumors, richly supplied with blood-vessels, supported by a very scanty framework, but abundantly supplied with an epithelial covering. In consequence of their very delicate construction, small portions of these growths are often torn off when the bladder is emptied, and are then, together with a certain amount of blood, voided with the urine.

Sarcoma of the bladder is a rare disease. It is encountered at long intervals in children.

Carcinoma of the urinary bladder is rarely seen as a primary growth, but it is often encountered as a tumor of secondary development. In the latter case the primary seat of the malignant growth is in women either in the body of the uterus, in its cervix, or in the vagina, while in men it is generally in the rectum. Primary carcinoma of the bladder has either a villous or a papillary structure (villous cancer), or that of a fungous growth, or else it presents the characteristics of an infiltration of the bladder-wall. In some instances it develops secondarily to stone in the bladder. Hæmaturia and catarrh are symptoms which often accompany new growths of the bladder.

FOREIGN BODIES AND CALCULI IN THE URINARY BLADDER.

Foreign bodies find their way into the bladder from neighboring organs; as, for example, by the breaking through of a dermoid cyst (hairs, bones, etc.) or of an extra-uterine fœtal sac. Here belong also those calculi which have come down from the pelvis of the kidney.

Stone in the bladder is a condition which develops chiefly in advanced life and almost exclusively in individuals of the male sex.

Calculi found in the urinary bladder are of two kinds: those which come from the pelvis of the kidney and consequently are of renal origin, and those which develop primarily in the bladder. The *calculi of renal origin* are apt to give rise to some form of inflammation, and, as a result of this, decomposition of the urine takes place, insufficiency of the bladder is

established, and the urine is only imperfectly voided. These calculi, after they have reached the bladder, increase in size through the formation of concretions upon their surface, and in this manner stones of considerable size are sometimes developed. Furthermore, calculi are formed independently in the bladder when retention of urine exists—a condition which is characterized by the fact that the bladder is no longer able to expel the urine completely. Such a condition of retention may develop in the course of some inflammatory trouble or a gonorrhœa, or it may result from hypertrophy of the prostate. In a similar manner concretions are formed around some foreign body, such as the fragment of a catheter which has been broken off in the bladder, or some other object which has—perhaps for masturbation purposes—been introduced into the urethra, and then has accidentally found its way into the bladder. Hairpins, cylindrical needle cases, and lead pencils are among the objects which have thus served as nuclei for the formation of a stone in the bladder.

These calculi in the urinary bladder are dangerous on account of the cystitis which accompanies them. Now and then a fatal termination occurs through pyelo-nephritis and septicæmia.

DISEASES OF THE URETHRA.

By far the most common and at the same time the most important disease of the urethra is *gonorrhœal inflammation* caused by the gonococcus. In addition to the alterations which are characteristic of all inflammations, it will be found, in the commencement

of the disease and during the acute stage, that the mucous membrane of the anterior portion of the urethra is coated with pus, and that individual pus corpuscles occupy the spaces between the flattened epithelial cells.   Later on, desquamation of the epithelium takes place, and small cells infiltrate the subepithelial tissues.   As a rule the inflammation does not extend beyond the superficial portions of the mucous membrane.   The gonococci are found chiefly within the cells, imprisoned in leucocytes and epithelial cells, and pus cocci are often mingled with them.

If the process becomes chronic in character (gleet), the pathological alterations will be found mainly in the membranous portion of the urethra.   They are as follows: Proliferation of the epithelium, substantial losses of tissue, and mere erosions, which often in the process of healing develop cicatrices and thus cause a narrowing of the urethra (strictures).   As further results of such strictures may be mentioned retention of urine, cystitis, hydronephrosis, and occasionally—in individuals of enfeebled constitution and advanced in years—pyelitis and pyelo-nephritis.

In women the gonorrhœal process is confined chiefly to the cervix uteri and the urethra, while the vagina, which is lined with an epithelium well adapted to resist the encroachments of the disease, participates in a more secondary fashion, through the irritation caused by the outflow of a purulent fluid from the uterus.   The inflammatory and infectious process may extend to the glands of Bartholin, the mucous membrane of the body of the uterus, the Fallopian tubes, the ovaries, and the peritoneum.   In men the disease may extend to the seminal vesicles, the pros-

tate, the epididymis, and the testicle. Through metastasis inflammation of the joints may be set up as a complication. Finally, in some instances, suppurative inflammation also develops in the connective tissue which surrounds the urethra, thus giving rise to *peri-* and *para-urethral abscesses*, the pus in some of these cases collecting in the preformed peri-urethral diverticula which are lined with epithelium. Some of these abscesses and also the secondary purulent inflammation of the inguinal glands (buboes) owe their origin to a mixed infection.

Injuries to the urethra (false passages resulting from the unskilful employment of the catheter) often give rise to an infiltration of pus and urine into the tissues at the root of the penis, or among those of the scrotum and perineum; and out of such infiltration severe forms of suppurative phlegmon, gangrene, and death through septicæmia may develop. (Plate 12.)

Sexual Apparatus.

DISEASES OF THE MALE GENITAL ORGANS.

Incomplete development of the testicles (hypoplasia, mikrorchia), associated with azoöspermia and sterility, is a defect of rare occurrence; and, besides, the individuals in whom it is encountered are often of normal development in all other respects.

Kryptorchismus is the result of the persistent failure of the testicles to descend from the abdominal cavity. The condition is one which occurs more often on one side (monorchismus) than on both, and it is also characterized by incomplete development of the testicle and by azoöspermia. The imperfectly developed testicle is generally to be found lying in the inguinal canal (inguinal testicle), and it is further distinguished by the fact that it is specially prone to be the seat of malignant growths, such as sarcoma and carcinoma.

Acquired atrophy of the testicle occurs in advanced life as a result of the compression and disturbed nutrition produced by hydrocele and by inguinal and scrotal hernias.

INFLAMMATION OF THE TESTICLE AND EPIDIDYMIS.
(Orchitis and Epididymitis.)

Inflammatory processes in the testicle are usually of a secondary character, the primary seat of the dis-

ease being in the prostate gland, the urethra, or the urinary bladder, from which regions the infectious inflammation spreads along the spermatic cord to the epididymis and testicle. It is particularly the gonorrhœal infection which spreads in this manner to the testicle, although other suppurative inflammations may also follow the same course. Metastatic infectious inflammations in the parenchyma of the testicle are of rare occurrence. They may develop, for example, in the course of a septico-pyæmia or during an attack of small-pox. Some of the milder forms of orchitis and epididymitis occasionally develop in a spontaneous fashion (that is, without any demonstrable cause); or they may be produced by a traumatism, in which case they usually terminate quickly in a return of the testicle to its normal condition or in more or less atrophy of the organ. As a result of these inflammatory processes the affected parts present a swollen and reddened aspect, and usually their consistence is increased. The exudation and infiltration of serum and cellular elements will be found both within the canaliculi and in the interstices of the tissues which lie between them. In the great majority of cases all this exuded material is absorbed and the organ returns to a fairly normal condition. It is a rare occurrence that an abscess forms and breaks first into the sac of the tunica vaginalis and then through the outer skin. Finally, cases are sometimes seen in which the epididymitis and orchitis have become chronic, as evidenced by the proliferation of the connective tissue of the epididymis and testicle and by the induration of the affected parts (chronic interstitial inflammation). At a more advanced stage of

terstitial orchitis atrophicans. They vary in size, their color is yellowish, they are dry, and their consistence is not unlike that of India rubber. Under favorable circumstances (as when the antisyphilitic treatment has proved successful) the inflammatory products are gradually absorbed and a fibrous cicatrix alone remains. An adhesive peri-orchitis, with obliteration of the sac of the tunica vaginalis, is very apt to be an accompaniment of syphilitic disease of the testicle.

TUMORS OF THE TESTICLE.

Among the primary tumors of the testicle sarcoma is the commonest. Adenoma and carcinoma occur more rarely. Tumors of a mixed composition are not uncommon. Such are cystoid growths, enchondromata, cysto-sarcomata, and cysto-carcinomata. These mixed tumors are of a teratoid nature, containing tissue-elements which have descended from the ecto-, meso-, and endo-derm. While they are of congenital origin, they lie dormant for years, and take on active growth only at some later period of life (after puberty). They are very closely related to the solid teratomata of the ovary.

The *scrotum* is often affected by elephantiasis, and also by a pronounced type of œdema, as in general dropsy.

Various inflammations, which may terminate in phlegmon or in putrefaction, may also involve the scrotum. Thus, for example, they may develop in the course of peri-urethral abscesses of the perineum, and as a result of the infiltration of urine resulting from defects of the urethra.

Among the *new growths* which are sometimes *seen in the scrotum*, the following may be mentioned: Lipomata and quite rarely carcinomata, particularly those remarkable (etiologically) and instructive cancers known as the tar and paraffin workers' and chimney sweepers' cancers. (Plate 14.)

DISEASES OF THE SPERMATIC CORD AND THE TUNICA VAGINALIS PROPRIA.

Inflammation of the spermatic ducts (*funiculitis*) may be excited by various causes. In rare cases an injury may be the cause of the trouble, and in that event an acute inflammation of the epididymis will scarcely fail to be an accompaniment. At first the parts are simply swollen and thickened, but later there may be suppuration and an abscess may form. In chronic (particularly syphilitic) orchitis and epididymitis the disease may also spread to the spermatic ducts, and as a result of this the connective-tissue walls of these channels and the tissues which envelop them undergo induration; and this in turn leads to closure (atresia) of the ducts.

By the term *hydrocele* is understood an accumulation of a watery fluid in the sac of the tunica vaginalis propria testis. This fluid is generally clear, but sometimes there is an admixture of blood, and the color may then be that of chocolate (*hæmatocele*). The term *congenital hydrocele* (h. congenita) is applied to that species of hydrocele in which the sac formed by the extension of the peritoneum down into the scrotum continues to remain open after birth. *Acquired hydrocele* (h. acquisita) is the commoner form

of the two. The conditions here are not those of a
simple accumulation of fluid in a sac, but rather
those of a diffuse inflammation (peri-orchitis serosa),
characterized by the pouring out of fluid into the in-
terstices of the tissues. The disease owes its origin
to some mechanical injury, or it may develop in con-
nection with an epididymitis gonorrhœica. In course
of time the serous exudation becomes absorbed and
the parts return to a natural condition; or connective-
tissue adhesions which cause an obliteration of the
sac of the tunica vaginalis may remain as a result of
the inflammation; or, finally, the inflammation may
become chronic in character (*hydrocele chronica*). In
the latter case the sac containing the fluid steadily
increases in size and assumes the shape of a pear.
It may increase to the size of a man's fist or even to
that of a child's head, and the imprisoned fluid may
weigh several pounds.

The term *varicocele* signifies a varicose enlargement
of the veins of the spermatic cord, in which condition
the enlarged veins form a convolution of worm-like
cords.

DISEASES OF THE PROSTATE.

Acute inflammation (*prostatitis*) with swelling of the
gland occurs frequently as a result of gonorrhœal ure-
thritis or in consequence of some mechanical injury
(employment of the catheter, false passages). If it
goes on to suppuration, abscesses are likely to form,
and these may either break or they may become en-
capsulated, their contents then undergoing inspissa-
tion and eventually becoming converted into a chalky
material. When a perforation occurs and a fistula is

formed, this may lead into the rectum, or into the urethra, or into the perineal region (rectal, urethral, perineal fistulæ).

Chronic inflammation of the prostate may result from a preceding acute inflammation. Under these circumstances the parenchyma of the gland presents a brownish discoloration. In chronic affections of the urinary apparatus which are of an infectious character abscesses are also likely to develop.

Tuberculosis of the prostate occurs as an intercurrent phenomenon in genito-urinary tuberculosis.

Enlargement of the prostate occurs with special frequency in advanced life—that is, in about one-third of all old men. In these cases the enlargement may be due either to a genuine hypertrophy or (more rarely) to the presence of a diffuse adenoma, or a fibro-myoma, or some mixed form of tumor (a sharp separation between the different forms of tumors being often an impossibility). The adenomata are generally characterized by a softer consistence, while the fibro-myomata are quite tough. The prostate is very rarely the seat of a malignant tumor (sarcoma or carcinoma). Enlargement of the prostate is apt to cause a narrowing of the prostatic part of the urethra or of the orifice of this canal in the bladder. The latter condition is observed particularly in those cases in which the middle lobe of the gland protrudes like a plug into the neck of the bladder, so that when the latter contracts it (the middle lobe) is forced like a sponge, under the pressure of the onflowing urine, against the orifice of the urethra. As a secondary result of this state of affairs there occur other alterations, like those which are observed in strictures of

the urethra. These are compensatory hypertrophy of the muscular walls of the bladder, dilatation of the ureters, hydronephrosis, and occasionally also a septic pyelo-nephritis, the issue of which is death.

Atrophy of the prostate is encountered in persons suffering from marasmus and in castrated individuals. It is also a fact, established by the results of recent operations, that a pathologically enlarged prostate—especially the softer variety—rapidly undergoes atrophy and diminution in size after castration. Experiences like these warrant the inference that the prostate, as an accessory sexual gland, is entirely dependent upon the integrity of the testicles, not only for its continuing existence but also for its development. When the nervous influence which binds these two glands together in close functional relationship is withdrawn, atrophy of the prostate begins. Resection of the seminal ducts acts upon the prostate in very much the same manner as does castration, but yet the effect is not so certain.

Concretions and calculi are often encountered in the prostate. The so-called concretions (which assume a bluish-violet color when treated with iodine, or a reddish hue, like the amyloid substances, when treated with methyl violet) are found quite regularly in the prostate of sexually mature men. They probably owe their formation to the coagulation of albuminous secretion or of cast-off cells. The calculi, which are usually present in considerable numbers, display a brownish or a yellowish color.

DISEASES OF THE FEMALE SEXUAL ORGANS.

OVARIES.

Defective development (hypoplasia) of these glands is observed as an accompaniment of rudimentary development of the uterus, the Graafian follicles being either entirely absent or else present in scanty number. This arrest of development is often associated with imperfect development of the entire body, as well as with other conditions, such as anæmia, chlorosis, abnormal smallness of the heart, and narrowness of the calibre and thinness of the walls of the aorta and larger arterial trunks.

Dislocation of the ovaries is often caused by the traction exerted by peritoneal adhesions (the result of adhesive and obliterating pelveo-peritonitis and peri-oöphoritis). Under these circumstances the ovaries are often completely embedded in such organized products of inflammation, and it is sometimes a difficult matter to discover where these organs are located, this being particularly the case when secondary atrophy has already taken place in them. Fixation of the Fallopian tubes in some abnormal position, obliteration of the abdominal orifices of these tubes, hydrosalpinx and pyosalpinx are among the common accompaniments of the condition described above.

Disturbances of the circulation (congestion, venous hyperæmia) owe their origin to the same causes which produce these conditions in other glandular organs. In addition to the physiological bleeding

which takes place when a follicle ruptures and a corpus luteum is formed, hemorrhages occur with comparative frequency in the cystic new growths of the ovary (formation of hemorrhagic cysts).

INFLAMMATION OF THE OVARY; OÖPHORITIS.

This is a rare process; it occurs more often as a secondary phenomenon than as an idiopathic manifestation.

In the *acute forms of the inflammation*—which may generally be divided into a follicular and an interstitial variety—the ovaries are swollen and highly œdematous. Scattered centres of suppuration (abscesses) are not often formed. An acute infectious oöphoritis occurs most frequently in connection with septic and purulent inflammations of the endometrium, Fallopian tubes, and pelvic peritoneum. In the latter case, if the patient recovers, adhesions will be found to have taken place between the ovaries and the neighboring organs, and ultimately the former will undergo atrophy.

Chronic interstitial oöphoritis (terminating in granular atrophy) is often observed as a result of debilitating constitutional diseases. It also develops as a result of unknown causes. Both ovaries are regularly affected by the disease. At first the inflammatory process, which is hyperplastic in character, attacks the cortex and gradually advances from here to the centre. In its progress the follicles are destroyed (through wasting) and consequently the functional activity of the gland ceases (in other words, sterility is established). Ultimately the ovaries will

be found in shrunken condition, with a consistence of board-like hardness and a surface rendered uneven by the presence of small nodules.

TUMORS OF THE OVARIES.

The ovaries are strikingly predisposed to become the seat of some form of pathological new growth. These are divided into solid tumors (from five to ten per cent of ovarian tumors) and cystic growths, into connective-tissue and epithelial tumors, and finally into benign and malignant growths.

1. *Connective-tissue tumors.* Chondromata are of very rare occurrence, while the fibroma (Fig. 1) is encountered somewhat more frequently. These fibrous tumors, especially the soft varieties, sometimes attain a great size — from twenty-five to thirty kilograms in weight. The

FIG. 1. — Myxo-Fibroma of the Ovary, with a Long Peduncle. (Preparation belonging to the Gynæcological Clinic of Munich.)

peduncle of the growth consists of the short and broad mesovarium. Smooth muscular fibres are also found scattered throughout the connective tissue (myofibroma), or portions of the tumor may show a chondromatous character.

2. *Among the solid tumors of the ovaries* the *sarco-mata* are the most frequently encountered. They vary in size from that of a hen's egg to that of a man's head, and often they involve both ovaries. Two varieties are distinguished: the spindle-cell and the round-cell sarcoma. These tumors possess a rather firm consistence and are of a whitish color. On section the cut surface presents a glistening and moist appearance. Quite recently cases of *angio-sarcoma* (endothelioma) of the ovaries have been reported. Formerly it was the custom to reckon among the cancerous growths those medullary sarcomata which are only rarely observed in children. Of the sarcomatous tumors found in the ovaries many are of a mixed character; as, for example, fibro-sarco-mata, adeno-sarcomata (from cystadenomata), and sarcoma carcinomatodes.

Secondary sarcomatosis of the ovaries is of rare occurrence. When this change takes place it is usually as a sequence to sarcomatosis and fibro-sarcomatosis of the peritoneum.

EPITHELIAL TUMORS.

Malignant tumors of epithelial origin are rarely observed in the ovaries. Now and then one of meta-static origin is seen. The scirrhous and the medul-lary varieties are the ones which are commonly observed in this organ, but in very rare instances a gelatinous cancer may be found. Generally the entire ovary is swallowed up in the tumor, and sometimes both ovaries completely disappear in this manner. It often happens that a carcinoma develops

from an adeno-cystoma of the ovary, and we may then term it a cystoma carcinomatosum or an adeno-cysto-carcinoma.

The *papillary epithelioma* (the papilloma of free surfaces) is also rarely encountered in the ovary. It consists of projecting villous growths, rich in blood-vessels and covered with an abundance of cylindrical epithelium.

The cystic tumors of the ovaries are seen in three different forms, viz., as simple follicular cysts, as multilocular cystomata or cystadenomata, and as dermoid cysts.

1. *Simple follicular cysts* (hydrops follicularis, dropsy of the Graafian follicles). In the early stages of the disease it is often still possible to discover the ovum. These cysts are either multiple or they may occur singly. As a rule they possess only one chamber. Their first development usually takes place at the age of sexual maturity. In the matter of size they differ greatly, some being no larger than the head of a pin, while others may attain the size

Fig. 2.—Multiple Cysts of the Ovary. (Oligocystic degeneration of the ovaries.)

of a man's head. On their inner surface these cysts are smooth, being lined with a single layer of cylindrical or flattened epithelium. The fluid contained within them is generally a clear serum, but now and then there may be an admixture of altered blood.

The *corpus-luteum cysts*, which develop from the corpus luteum and sometimes reach the size of an apple, constitute a special subdivision of the simple follicular cysts. They contain either a watery or a syrup-like fluid of a yellowish or a reddish-brown color, and on their inner surface, which presents the color of rust or of ochre yellow, the formation of well-defined folds can usually be made out.

2. *Multilocular cystomata or cystadenomata* (cystoid growths) develop as a rule from the germ-epithelium

FIG. 3.—Proliferating Papillary Cystoma of the Ovary. The cyst wall contains aggregations of papillary formations which at one point proliferate through the wall. (The preparation belongs to the Munich Gynæcological Clinic.)

or from Pflüger's tubes, and they are among the commonest of ovarian tumors. In size they differ very greatly, according to the stage of development which they have reached. They may weigh as much as twenty, thirty, or even forty kilograms. The
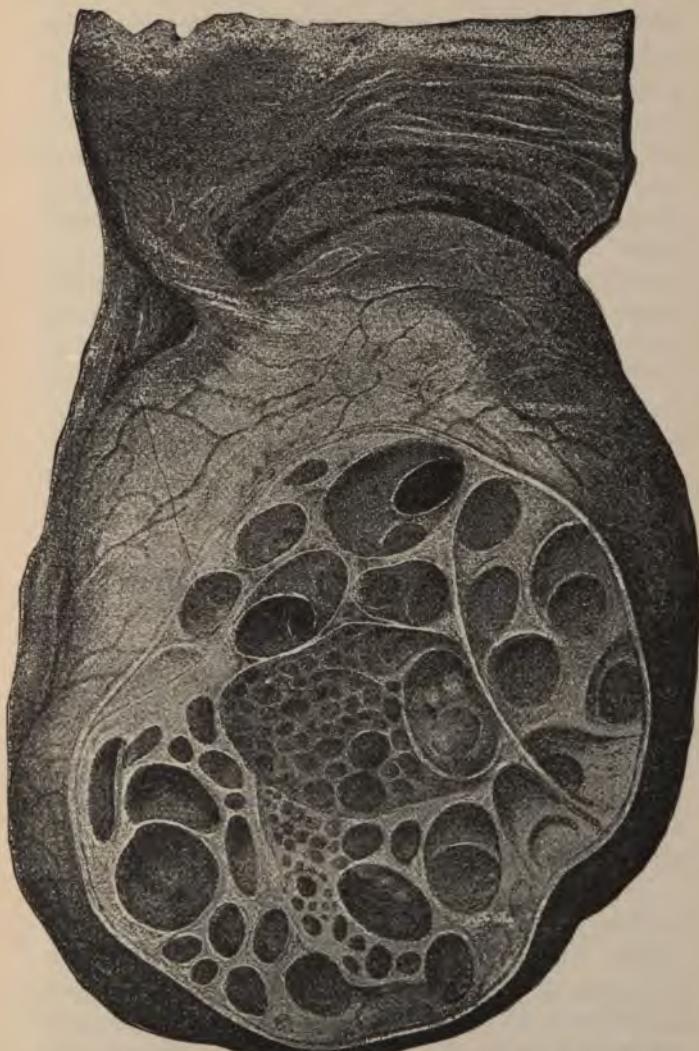
Fig. 4.—Multilocular Glandular Myxoid Cystoma of the Ovary, with Twisting of the Peduncle. (Preparation belonging to the Munich Gynæcological Clinic.)

adeno-cystomata are of two varieties: *the glandular* and *the papillary*. These tumors, which have a smooth surface, are of a somewhat soft consistence, and when palpated they seem to fluctuate. When cut open these tumors afford escape to grayish and grayish-white masses of a glairy and tough mucus, often mixed with altered blood (the result of twisting of the peduncle). Substances resembling mucin (pseudo-mucin) are present in abundance in these masses. The surface of a section of the tumor made with the knife shows plainly the irregular, honeycomb-like construction, for it brings into view a number of large and small cysts, placed side by side and communicating one with another at numerous points. Whereas the *glandular* tumors (Fig. 4) possess a smooth internal surface, and are characterized by the fact that new cysts are formed by the folding in and subsequent constriction (at the starting-point) of a portion of the epithelial layer, the *papillary cystadenomata* (Fig. 3) present, in the chambers of which they are composed, the phenomenon of an abundant new production of villous and papillary structures covered with cylindrical and ciliated epithelium.

The papillary cystadenomata (cystoma proliferum papillare) often involve both sides. They are generally located within the broad ligament, and their growth is quite slow.

Mixed forms of tumors which have developed from multilocular cystadenomata are of frequent occurrence. Thus, for example, a carcinoma sometimes develops in what was originally a cystadenoma. Cystadenomata involve both ovaries in only five per cent of all cases. In the majority of instances (sev-

enty per cent) cystadenomata develop in women who have not yet reached the age of forty, while the number of instances in which the disease has been found between the ages of forty and fifty amounts to only twenty per cent. The disease is comparatively rare in advanced life.

3. *Dermoid cysts* of the ovaries constitute between three and four per cent of all ovarian tumors. They belong in fact to the teratoid tumors. They are usually of moderate size; they grow slowly, and they are of congenital origin. The inner wall of these cysts resembles in structure the external skin. The tissue elements present are epidermis, hairs, hair follicles, and both sebaceous and sweat glands. Small projecting nodules (rudimentary cephalic caps) are also observed. These contain portions of bone with teeth, and often the rudiments of brain substance, eyes, and ciliated endoderm. The rudiments of the circulatory apparatus (heart, blood-vessels) or of the kidneys are never found. The conditions observed here, therefore, do not indicate displaced embryonal germs, but rather parasitic fœtuses, or rudimentary ovarian parasites, or the abortive malformation of an egg cell.

The free contents of the cysts consist of a sebaceous, thickish material in which numerous hairs are embedded.

DISEASES OF THE FALLOPIAN TUBES.

Displacements of the Fallopian tubes, especially in a backward and downward direction, are often associated with bending, dragging, or obliteration of the

same. They may be due to various causes, such as changes in the position of the uterus and ovaries, and connective-tissue adhesions in the true pelvis (adhesive pelveo-peritonitis).

Narrowing and closure of the tubes are often the result of catarrhal changes, and also of closure of the abdominal opening through peritonitic adhesions. At the uterine orifice the tube may become narrowed by the encroachment of a tumor of the uterus. Finally, changes in the position of the tube itself may cause bending of the same and a narrowing of its calibre.

HYDROSALPINX.

(Plate 20.)

As a result of closure of the abdominal orifice the secretions in the tube accumulate and cause dilatation first of the lateral portion and then gradually of the entire tube. Thus dilated the tube becomes filled with a clear, watery fluid (retention dropsy), and may attain the size of a man's finger or thumb. It also, as a result of the dilatation, assumes the shape of an elongated tortuous sac, not unlike a sausage or a portion of the intestines. Furthermore, owing to the presence of transverse folds which project from the walls of the tube into its interior, there will be formed a series of cavities, which follow one after the other in a row, like the beads of a rosary. When hydrosalpinx develops on both sides, sterility is sure to be the result.

HEMORRHAGE FROM THE MUCOUS MEMBRANE OF THE FALLOPIAN TUBES.

This may result from a variety of causes, such as menstruation, inflammatory processes, and infectious diseases. The blood which is thus poured out may escape into the abdominal cavity and there form a *retro-uterine hæmatocele*. But if the abdominal orifice is obliterated the escaping blood will accumulate in the lumen of the tube, thus causing it to become dilated. To this condition, which, when seen from without, presents the same appearances as does a hydrosalpinx, the name *hæmato-salpinx*, or *hæmatoma of the Fallopian tube*, is given. Finally, a hæmato-salpinx may be the result of a gynatresia (atresia of the cervix or of the vagina), and with this condition a hæmatometra will be associated.

In tubal pregnancy a hemorrhage, which is often fatal, may be expected to occur when the sac ruptures.

INFLAMMATION OF THE FALLOPIAN TUBES; SALPINGITIS.

Inflammation of the tubal mucous membrane may occur in either an acute or a chronic form. As a rule the inflammatory and infectious process travels in a directly continuous manner from the mucous membrane of the uterus.

The milder forms are termed *tubal catarrh* (salpingitis catarrhalis, in which disease the pathological changes are those of a mild inflammation of the mucous membrane, accompanied by a muco-serous secretion. Through an extension of the disease to the

abdominal orifice a limited peritonitis may be excited, or the inflamed tube may itself become closed, and dropsy from retention may follow. Complete restoration of the parts to a healthy state is a common result.

In the *cases of severe inflammation*, particularly those in which a gonorrhœal inflammation—both with and without other infectious elements—has spread from the vagina and the uterus, the exudation assumes a purulent character. Pus accumulates in large quantity, and to this condition the term *pyosalpinx* (Plate 23) is applied. When the peritoneum is involved at the same time, the condition is spoken of as a purulent salpingo-peritonitis and perisalpingitis. A purulent breaking down of the tubal wall and perforation into the abdominal cavity are events which occur only rarely. After the purulent salpingitis has lasted for a long time, the parts generally return to a healthy condition.

A salpingitis with a secretion of ichorous pus, or of a pus which is septic, occurs now and then as a result of a puerperal infectious endometritis, and may also terminate in a fatal *salpingo-peritonitis.* More commonly the puerperal endometritis spreads to the peritoneum by way of the lymph channels of the uterus and its appendages. It is a rare thing during the puerperal condition to see an acute purulent peritonitis develop, after a latent stage of a certain duration, from a purulent salpingitis (late infection).

Tuberculosis of the Fallopian tubes, like that which involves the mucous membrane of the uterus, is observed in the form of a cheesy inflammation. The

tubes are converted into tough and rigid structures, and the mucous membrane presents the appearance of being fissured and in a state of cheesy necrosis. The process can travel in a downward direction, starting from the abdominal orifice (as an extension of tuberculous disease of the peritoneum), or it may travel upward along the tube from the uterus as a starting-point.

New growths of the Fallopian tubes are extremely rare. Those hitherto seen were fibromata, myomata, and papillomata.

TUBAL PREGNANCY.

(Plate 22.)

The so-called extra-uterine pregnancy (ectopic pregnancy) occurs in a variety of forms. They are as follows:

1. *Abdominal pregnancy* (graviditas abdominalis), of which there are two varieties—the peritoneal and the extraperitoneal. The fructified egg may develop in any part of the peritoneum or between the layers of the ovarian ligament, close to the hilus of the ovary.

2. *Ovarian pregnancy* (graviditas ovarialis), in which the development of the egg takes place upon the ovary.

3. *Tubal pregnancy* (graviditas tubaria). When the ovum develops in the commencement of the tube (the uterine portion), the condition is termed an interstitial or tubo-uterine pregnancy (a very rare variety). In a second form the ovum develops in the free part of the tube; and to this the term tubal preg-

nancy, in its narrower sense, is applied. The third variety, which is termed tubo-abdominal pregnancy, is of very great rarity; for, as a matter of fact, the cases which are generally believed to belong in this category are really cases of misinterpreted tubal or abdominal pregnancy.

Very often—perhaps even in a majority of instances —the abdominal and ovarian varieties commence originally as a tubal pregnancy.

In the majority of cases (seventy per cent) of extra-uterine (ectopic) pregnancy either a tubal abortion (that is, an expulsion of the ovum through the abdominal mouth of the Fallopian tube) or a rupture of the fœtal sac takes place during the first three months. Various results may follow: as, for example, the death of the embryo and the absorption of its component elements; the formation of a hæmatoma behind the uterus (retro-uterine hæmatocele) when the rupture takes place into the abdominal cavity, or the development of this same lesion in the substance of the broad ligament when the hemorrhage occurs at this point; or a fatal issue may result from internal hemorrhage; or the embryo may go on developing up to full maturity of fœtal life, and may then be delivered by means of operative interference; or the embryo may die and become converted into a lithopædion; or, finally, suppuration may take place in the fœtal sac, and the abscess may break into some neighboring organ (bladder or intestine).

DISEASES OF THE UTERUS.

CHANGES IN POSITION.

The most important changes in position are those which are due to *versions*—displacement of the organ as a whole—and those which are due to its being bent upon itself—*flexions*. In the case of the former of these changes the angle where the bending occurs is situated in the vault of the vagina, the entire uterus being displaced forward or backward; while in that of the flexions the angle of bending lies very near the os internum of the uterus—in other words, the axis of the body of the organ forms an acute or a right angle with the axis of its cervix.

Flexions are either movable or fixed, in the latter case through the instrumentality of the connective-tissue products of an inflammation.

Anteflexion of the Uterus.—In this condition the body of the organ rests upon the anterior part of the vault of the vagina, the bent portion either being angular or presenting the shape of a retort. The movable variety of anteflexion may, as a rule, be considered as a physiological condition, while the fixed variety, which is of rare occurrence, is of pathological origin.

Anteversion of the uterus is a condition in which the axis of the cervix and of the body of the organ lies in a horizontal direction, at right angles to the transverse axis. The entire uterus having fallen forward, its external orifice looks backward toward the sacrum. When increased intra-abdominal pressure continues for a long time—which may happen, for example, in

kypho-scoliosis accompanied by marked shortening
of the spinal column—a pathological movable ante-
version may result.   The author has observed a num-
ber of instances in which this pathological condition
existed.

Retroflexion of the Uterus.—In this condition the
body of the uterus is bent backward, in such a man-
ner that it forms a right angle with the cervix, while
the fundus sinks down into Douglas' cul-de-sac.
The point where the bending occurs is in close prox-
imity to the os internum.   The movable variety of
retroflexion of the uterus produces as a rule insignifi-
cant symptoms; while, on the other hand, the adhe-
sive or fixed form—that in which the organ is bound
down by connective-tissue pseudo-membranes—is of a
more serious nature.   This form of retroflexion is par-
ticularly apt to occur as a sequel to childbirth, when
the uterus is large and heavy and the tissues in the
neighborhood of the os internum are relaxed.   Further-
more, it also sometimes occurs (with or without de-
scent of the organ) when excessive abdominal pressure
is indulged in too often, and when intestinal hernia
develops after the occurrence of numerous labors.

Retroversion of the uterus is a condition in which the
fundus of this organ lies as if it had fallen over back-
ward, while the outer orifice (os externum) looks
toward the symphysis pubis.

Descent and Prolapse of the Uterus.

In simple *descent* the uterus occupies a lower posi-
tion than it normally does; the highest part of the
vagina being folded in, and the os externum lying in
the space between the vulvæ.

The *falling down* (or *prolapse*) *of the uterus* is said to be *incomplete* when the lower part of the organ can be seen lying between the vulvæ.

The condition can be termed a *complete prolapse* when the entire uterus lies like a tumor in front of the vaginal opening, in which condition the everted vaginal wall covers the prolapsed organ.

The prolapsed uterus is generally much enlarged, especially in its lower segment, and at the same time it is markedly engorged with blood by reason of interference with its venous circulation. The vaginal mucous membrane which covers the organ is invariably thickened, and the outer layer of epithelium is converted into something closely resembling an epidermis. The cavity of the uterus seems to be enlarged, and its lining mucous membrane is decidedly hyperæmic. Among the secondary results may be mentioned decided stretching of the urinary bladder and a diverticulum-like protrusion of both it and the rectum (cystocele and rectocele). The orifice of the urethra is usually visible on the anterior aspect of the prolapsed parts. Among the secondary results of such a prolapse may be mentioned retention of urine, catarrh of the bladder, and a tendency to the formation of calculi. When the parts are examined from behind (*i.e.*, through an opening in the abdominal walls) there will be seen, between the bladder and the rectum, a funnel-shaped depression into which the ovaries and tubes have been pulled from above downward. Various factors play a part in *the development of prolapse of the uterus*, and of these the most important are the two following: a relaxed condition of the pelvic viscera, by reason of too frequent pregnancies,

and an abnormally heavy uterus, both of which factors are present during the lying-in period (only from six to seven per cent of all cases occurring in women who have borne no children). Among the other factors which are occasionally present may be mentioned the following: increased intra-abdominal pressure, such as takes place when some task requiring heavy bodily labor is performed (lifting a heavy load, a jump, a fall). In advanced life the disappearance of a certain amount of fat from the body is likely to favor this relaxed condition of the pelvic organs. Still other favoring factors are these: abnormal breadth of the pelvis and slight inclination forward of its vertical axis, and extensive ruptures of the perineum.

Inversion of the uterus occurs both during the lying-in period and also at other times. In the former case the condition is produced when a pulling-down force is applied to that relaxed and thin portion of the uterus where the placenta was attached. Outside of the lying-in period, the condition may be produced by pedunculated tumors (submucous myomata) which tend to advance farther and farther downward, and at the same time to exert traction upon a portion of the uterine wall. In this way, a complete inversion of the uterus will in due time be produced, the inverted parts eventually presenting themselves outside the vulvar opening.

STENOSIS AND ATRESIA OF THE UTERUS.

Congenital narrowness of the genital canal involves as a rule the neck of the uterus throughout its entire length, and particularly the outer orifice of the os ex-

ternum. In these cases a very narrow opening may be seen at the end of the hypertrophied, tough, and usually somewhat pointed or cone-shaped portio vaginalis of the uterus.

Acquired stenosis and atresia of both the os internum and the os externum occur now and then as a result of some traumatism produced during labor or in consequence of an inflammation which has developed during the puerperal period. The condition may also result from the presence of a flexion or a new growth. In women who have never borne children, stenosis and atresia may result from a growing together of opposite mucous surfaces which are to a greater or less extent eroded, or from an obliteration of the cervical canal through the growth of cicatricial tissue which has followed the employment of cauterization. Among the results of such narrowing or closure may be enumerated the following symptoms: discomfort or actual pain (dysmenorrhœa) during menstruation, and retention of the menstrual blood (hæmatometra, hæmatosalpinx), and, in older women, the accumulation of a sero-mucous secretion (hydrometra) or of one which is purulent in character (pyometra), the latter condition developing in the course of some inflammatory process of an infectious nature.

HEMORRHAGE FROM THE UTERUS.

A distinction must be made between hemorrhages which take place into the cavity of the uterus and those which occur outside in its immediate vicinity.

Hemorrhages of the non-pregnant uterus are con-

nected in some way either with menstruation or with a variety of pathological conditions.

The increased flow of blood during the menstrual period (when it reaches the point at which it may be spoken of as a menorrhagia) is often due to constitutional causes (such as a hemorrhagic diathesis, or one of the infectious or toxic general diseases), or it may be dependent upon some local disease of the uterine mucous membrane (hemorrhagic endometritis, or some form of new growth, like a submucous myoma, a mucous polypus, or a carcinoma).

Hemorrhages outside the uterus, in its immediate vicinity, are either intraperitoneal or subperitoneal, or else they combine both characteristics. Intraperitoneal hemorrhages which take place into Douglas' space and which often, after coagulation has taken place, become encapsulated, constitute tumors of considerable size (pelvic hæmatoma, retro-uterine or peri-uterine hæmatocele, Plate 22). The blood which is thus poured out comes from the ovaries, from follicular cysts which have burst, or from the Fallopian tubes (rupture of a hæmatosalpinx, a tubal pregnancy). Finally, a proliferative pelveo-peritonitis, when associated with the new formation of blood-vessels, may take on a hemorrhagic character and lead to the formation of an inflammatory hæmatoma. Such accumulations of blood generally undergo absorption, and, when this happens, a rusty brown or a slate-colored discoloration of the pelvic peritoneum remains behind. It is only in rare cases that the mass breaks down into pus, which then finds an outlet either into the rectum or into the vagina, and that a diffuse fatal peritonitis is set up.

RUPTURE OF THE UTERUS.

Rupture of the pregnant uterus is an accident which often happens during labor, in cases in which there are obstacles that tend to prevent the birth of the child. This may happen, for example, when the fœtus is too large for the channels through which it must pass. In this event the tissues of the cervix get jammed in between the fœtus and the wall of the pelvis, and it is here that the rupture eventually takes place. Almost invariably (ninety per cent of all cases) the rupture results from one of two things—either the bony pelvis is narrowed* and thus presents an obstacle to the birth of the child, or the os uteri is composed of such unyielding tissues or has such a narrow canal that it cannot dilate in a normal manner.

The seat of the rupture is generally in the cervix, and often on the left side. The line of the tear usually runs in a longitudinal direction.

Shallow tears in the mucous membrane are of common occurrence in childbirth.

* A rupture rarely results from the pressure produced by excessive labor pains. Normal contractions occasionally induce an excessive stretching of the cervix and lower segment of the uterus, and they may even produce a rupture when coincidentally an induration, stenosis, or atresia of the cervix exists—more rarely, when the vagina is the seat of one of these pathological conditions. Then, besides, markedly thinned and very yielding spots in the walls of the uterus—spots indicative of the harmful effects of previous severe labors—are a predisposing factor in the development of a rupture. The same causes may lead to rupture of the vault of the vagina in a case in which the os uteri has been drawn upward and the genital canal has been exposed to excessive stretching in a longitudinal direction.

A *rupture* is said to be *complete* when the tear extends through all the layers, and *incomplete* when it extends only part way through the wall of the uterus. The relation between the complete and the incomplete is as 100 to 8. Among the secondary results may be mentioned subperitoneal hemorrhage and the pouring out of blood into the abdominal cavity. In addition to the spontaneous ruptures there are those which are due to the employment of violent therapeutic measures, both manual and instrumental, and those which result from some mechanical force acting from without, such as a fall, a blow, etc. These latter occur rarely.

Healing and cicatrization of the ruptured uterus are results which do not often take place. Operative interference now and then brings about this favorable result. In the majority of cases (ninety-five per cent), however, death soon follows, either as a result of hemorrhage or through secondary septic infection. Rupture of the uterus occurs about seven times oftener in women who have already previously given birth to children than among those who are passing through their first labor.

ATROPHY OF THE UTERUS.

A physiological atrophy of the uterus—if we except the puerperal involution of this organ—occurs only after the menopause has been reached. In this condition the uterus shows a diminution in size in all its diameters, and a marked shrinking of the vaginal portion. After the operation of spaying, and also as a result of inflammatory granular atrophy (sclero-

sis) of the ovaries, the following changes take place in the uterus: diminution of its volume and weight, disappearance of the ciliated epithelium and its replacement by an epithelium composed of cubical cells, and finally the glandular elements diminish in number and in part become converted into small cysts. A genuine pathological and pre-senile form of atrophy of the uterus can develop from unknown causes (excessive lactation?) during the puerperal period. In this form the organ will be found to have extremely thin walls, which may easily be perforated, while the vaginal portion projects like a small plug into the vagina.

INFLAMMATION OF THE UTERUS.

According to the regions in which the inflammation is localized it is customary to distinguish the following varieties: endometritis, metritis, perimetritis, and parametritis.

Endometritis.

This disease, which is one of the commonest to which the female sex is liable, is encountered in various forms and degrees of severity. Thus, for example, in accordance with the duration, we may distinguish an acute, a subacute, and a chronic form; in accordance with the underlying cause, an infectious and a non-infectious form, a bacterial and a non-bacterial form; and, finally, according to the nature of the exudation and the character of the inflammatory products, an exudative and a proliferative (or productive) form. There are also a puerperal and a non-puerperal variety of endometritis.

If the body of the uterus is the part chiefly affected, it is customary, in describing the disease, to employ the term *endometritis corporis*, while that of *endometritis cervicalis* should be employed when the disease is confined mainly to the cervical portion. Both of these varieties of endometritis are often present, in a given case, at one and the same moment of time, and it also often happens that the disease passes from one form into the other. From an anatomical standpoint it is preferable to lay stress upon the distinction between the acute and the chronic forms of the disease.

Acute Endometritis.

The causes which often lead to the development of an acute endometritis are the following: the gonococci and certain pus exciting micro-organisms (streptococcus and staphylococcus) which are present either during the lying-in period—at which time the bacteria of decomposition are often associated with them (ichorous puerperal endometritis)—or after certain operative procedures upon the uterus (septic endometritis of traumatic origin). Milder forms of acute endometritis (endometritis catarrhalis) are encountered in the course of certain infectious diseases. The most important and the most dangerous form of this disease is—

Puerperal Septic Endometritis.
(Plate 16.)

It may develop after labor at full term and also after a miscarriage—both with and without mechanical lesions. While hyperæmia and well-marked œdema are certainly predisposing factors, of greater

importance are tears and bruises of the mucous membrane, inasmuch as these serve as points of entrance for the septic micro-organisms, which invade the tissues and multiply within them. This is particularly apt to happen after difficult labors (contracted pelvis, instrumental delivery), and also in those cases in which still-adherent remains of the placenta and fœtal membranes, or tears of the cervical portion of the uterus, offer favorable points where the agents which give rise to infection may effect a lodgement. After the initial stages of the inflammation (swelling, hyperæmia, and increased secretion of a fluid mixed with blood) have quickly passed away, the mucous membrane—in the more serious and the fatal cases—seems to undergo a conversion into a dirty gray or a grayish-yellow mass of the consistence of tinder. This change is especially noticeable at the points where the placenta had been attached or where a tear had occurred, at which places discolored eschars may be seen. The contents of the uterus consist of a bad-smelling, abnormal-looking fluid, composed of altered blood, pus, and cast-off masses of dead tissue. As a rule, those cases which end in death manifest a tendency, on the part of the uterine tissues, to undergo necrotic changes. We may speak of them as cases of ichorous endometritis, a disease in which well-marked putrefactive changes may, in some instances, be observed in the uterus. As is true of septic and infectious processes generally, there is a tendency in puerperal endometritis to spread to neighboring parts. In some cases the disease spreads by way of the lymph spaces (as in the case of erysipelatous and phlegmonous metritis), while in others it follows

the course of the larger lymph channels, giving rise
to *puerperal metrolymphangitis*, in which disease the
lymph vessels which run alongside the cervix, where
the broad ligaments are attached, are filled with pus.
In still other cases the connecting link between the
local infection and the secondary septico-pyæmia
is furnished by infectious thrombi which are con-
tained in the veins of the uterine walls (the result of
puerperal metrophlebitis) and which send extensions
into the pampiniform plexus of veins and into the
internal spermatic vein. Among the *secondary proc-
esses* there remains to be mentioned *purulent perito-
nitis.* This often develops quite rapidly from a septic
endometritis, the micro-organisms which cause the
infection reaching the peritoneum from the mucous
membrane of the uterus either by way of the larger
lymph channels--which are also at the same time in-
flamed and filled with pus—or (more rarely) by direct
continuity along the Fallopian tubes (puerperal sal-
pingitis), or finally by some direct route (not macro-
scopically demonstrable) from the internal surface of
the uterus to the peritoneum. In the latter event it
is possible, and especially if antiseptic irrigations of
the cavity of the uterus have been carried out, for
the primary process in the mucous membrane of the
uterus to run its course almost without leaving a
trace behind; and when this happens, therefore,
nothing will be found at the post-mortem examina-
tion beyond the secondary purulent peritonitis.

In the majority of cases we may be sure that we
are dealing with a genuine septic infection due to the
penetration of pathogenic germs (*streptococci*) into
the tissues, while in the minority there may be a

putrid intoxication with the products of decomposition, or else there may be a combination of both processes—infection and intoxication.

Infection of the puerperal uterus may usually be attributed to *infection introduced from without* (direct or indirect transfer of the contagium), as by means of unclean hands or instruments,* and only exceptionally to that produced by pathogenic germs which were already previously present in the vagina or at the vaginal entrance (cases of so-called self-infection).

In the normal secretions of the genital organs of healthy pregnant women streptococci are not found. As a matter of fact, these secretions, through the bactericidal power which they possess, furnish protection against pathogenic germs which sometimes (as saprophytes) make their appearance when certain changes take place in these secretions. When the processes which take place at childbirth and during the lying-in period put a temporary stop to this natural immunity, these saprophytes may for a certain time assume a parasitic character.

Gonorrhœal endometritis shows a decided tendency to localize itself in the neck of the uterus (gonorrhœal cervical endometritis), and the mucous membrane of the body of the organ may escape entirely. The pathological alterations are hyperæmia and redness

* Out of 149 cases of puerperal fever which came under observation between the years 1877 and 1883, in the city of Basle, 44 (= 30 per cent) passed through the hands of two midwives. *A propos* of this subject, it is of importance to note the fact that infection of the genital canal takes place both before and after the birth of the child, through contact of the fingers with the parts—through vaginal infection (due to too much intermeddling).

of the vaginal portion, swelling of the cervical mucous membrane, and the escape of a purulent secretion from the orifice of the cervical canal. In some cases, when the mucous membrane of the body of the uterus becomes involved, an analogous ascending inflammation of the Fallopian tubes and pelvic peritoneum follows in course of time.

The disease may disappear entirely or it may persist in a chronic form.

In *chronic gonorrhœal endometritis* the fluid secreted becomes more and more scanty, looks like milk, and is sometimes glassy in appearance. Gonococci are generally very few in number and difficult to find.

Chronic Endometritis.

According as the framework or the gland tubules are the structures chiefly involved, two varieties of this disease are usually recognized—an *interstitial* and a *glandular endometritis*. In the interstitial variety the connective-tissue stroma is both hyperæmic and œdematous; its tissues are in a proliferating state, and cellular elements fill its interstices; and generally this condition is associated with a discharge of a purulent fluid from the surface of the mucous membrane. In glandular endometritis the glands appear to be increased in number, to have grown longer, and to have assumed a serpentine shape. This is particularly true of cases in which a true dysmenorrhœa exists.

In the higher degrees of the disease the mucous membrane appears to have undergone considerable thickening, its surface being thrown into rounded hummocks and ridges. To this condition the terms

fungous endometritis, hypertrophic endometritis, and *proliferating endometritis* are applied. When polypoid growths spring from this thickened mucous membrane, it is customary in speaking of the condition to apply to it the term *endometritis polyposa.* Polypi of this character are often pedunculate, and they may contain small or even quite large mucous cysts, which have originated through the obliteration of the mouths of glands and the retention of their secretion. These fungous forms of chronic endometritis which are associated with catarrhal symptoms are usually not of bacterial origin; germs are not found in them.

Both in the acute and in the chronic forms of *cervical endometritis* it very often happens that *erosions* develop upon the vaginal portion of the uterus. The picture presented under these circumstances is that of a mucous membrane very much reddened in the vicinity of the external orifice of the uterus, having an uneven and somewhat excoriated surface, and presenting some actual ulcerations of slight depth. Papillary excrescences (as well as papillary erosions) and follicular ulcerations are also among the lesions which are sometimes observed. As a result of inflammatory swelling of the cervical mucous membrane the outer orifice of the uterus protrudes to a greater or less extent (*ectropium*).

Chronic endometritis often develops from a gonorrhœal infection, or the cause may be found in pus cocci, and this is very apt to be the case in the interstitial variety of the disease. The latter may also develop as a sequel to birth at term or a miscarriage; or it may follow some operative interference; or,

finally, it may represent the outcome of a secondary infection, which has developed in consequence of an acute gonorrhœa.

Fetid extrapuerperal endometritis, which is associated with the pouring out of an exudation of ichorous pus, develops now and then in elderly women.

TUBERCULOSIS OF THE UTERUS.
(Plate 18.)

Tuberculosis of the uterus rarely occurs as a primary disease, but it is quite often encountered as a secondary manifestation, in association with some other disease—generally with tuberculosis of the Fallopian tubes.   The tuberculous infection of the female genital tract occurs either through some external agency (as from connection with a man who is suffering from tuberculosis, and especially from tuberculous disease of the genito-urinary organs, or from an external self-infection) or by an extension of the disease from the Fallopian tubes (peritoneum), or else by way of the blood circulation.   The *genital tuberculosis of women* is usually a descending disease; that is, it extends downward from the Fallopian tubes to the mucous membrane of the uterus.   Whereas in the male sex the genital tuberculosis is very often associated with tuberculous disease of the urinary apparatus, in women the two almost never occur together.

As seen in actual practice tuberculosis of the uterus usually presents itself in the form of a rather sluggish cheesy inflammation of the mucous membrane (subacute or chronic cheesy endometritis).   At the commencement the internal surface of the uterus is cov-

ered with small, yellowish, superficial, sinuous ulcers. At a more advanced stage it will be found to have undergone a change into an opaque yellow, crumbling, friable, fissured mass, in the midst of which a few miliary and even somewhat larger yellowish tubercles may be seen. In addition to this diffuse cheesy infiltration of the mucous membrane, miliary nodules, and occasionally also papillary tuberculous vegetations, are also encountered—particularly in the cervix uteri—in exceptional cases. Owing to the retention of these products of inflammation intermingled with pus, the cavity of the uterus becomes dilated (pyometra), and often at the same time other pathological conditions will be found to be present; such as, for example, tuberculosis of the Fallopian tubes and pyosalpinx, tuberculosis of the pelvic peritoneum, and intestinal tuberculosis.

PERIMETRITIS; PELVEO-PERITONITIS.

(Plate 23.)

This disease either appears as a part of a diffuse peritonitis, in which the exudation and the local alterations of the serous membrane are generally most pronounced in the pelvic peritoneum, or else the inflammation develops out of some disease of the vagina, the uterus, or the Fallopian tubes, and more particularly in connection with some gonorrhœal or septic puerperal process involving these pelvic organs. The inflammation of the serous membrane runs an acute, a subacute, or a chronic course. The exudation varies in character, being at one time an opaque serum (partly serum and partly cellular elements), at

another a purulent or a fibrino-purulent fluid, and at still another a blood-stained fluid; only rarely—that is, when a communication exists between the peritoneal cavity and some neighboring organ—is the exudation an ichorous pus. The anatomical alterations are essentially the same as those observed in general peritonitis. Under the most favorable conditions, and especially in the milder forms of the disease, the inflammatory products are absorbed and an incomplete return of the parts to a normal condition follows; that is, adhesions form and a partial obliteration of the pelvic cavity takes place (*adhesive pelveo-peritonitis*). As a result of this condition there are apt to be dislocations of the ovaries and Fallopian tubes, and also flexions of the latter. The organized pseudo-membranes which have formed during such an inflammation, are at one time as delicate as a cobweb and easily torn, at another quite tough. The lining peritoneum is also often thickened and in places presents a lumpy appearance. In other cases the exudation becomes inspissated, and separate sacculated foci of pus are formed (intraperitoneal pelvic abscesses, retro-uterine pyocele). At such spots doughy swellings will be found, and fistulæ are likely to follow their formation; the routes pursued by these fistulæ following either an outward direction, through the skin, or one leading into one of the hollow viscera of the smaller pelvis (the intestine or the bladder).

In *extraperitoneal* or *retroperitoneal suppuration* the term *parametritis* is employed when the inflammation localizes itself in the connective tissue in the neighborhood of the ligamenta lata and the vaginal portion of the uterus. The inflammatory process

produces an exudation which is either watery (a
cloudy serum), purulent, or—when it has lasted for
a sufficient length of time—plastic in character, in
the latter case developing into a connective tissue
which tends to shrink and to form callosities (scleros-
ing parametritis). Parametritic abscesses may also
break through into adjacent organs (the intestine, for
example). The acute exudative forms are generally
septic in character, the result of puerperal infection,
or else they develop in consequence of ulcerations or
mechanical injuries of the vagina, of the vaginal por-
tion of the uterus, of the cervix, or even of the rectum.

NEW GROWTHS OF THE UTERUS.

Myoma, Leiomyoma, Fibromyoma.

(Plate 20.)

The myoma, an extremely common homologous
new growth of the uterus, is encountered in from ten
to twelve per cent of all female cadavers, and most
frequently (two-thirds of all the cases) in women be-
tween the ages of thirty and fifty. Tumors of this
nature consist of smooth muscular fibres and connec-
tive tissue, and they vary greatly in size, some being
no larger than a hempseed, while others attain the
size of a man's fist or even of his head. They have
a rounded form (globular myoma), are separated from
the surrounding tissues by sharply defined limits,
and are often multiple in character. They have a
somewhat whitish color, are very tough, and creak
under the knife. The fundus uteri and the posterior
portion of the body of this organ are the favorite

seats of the growth, while the cervix is only rarely involved. According to the situation occupied by the tumor it is customary to distinguish three different varieties, viz., the subserous or subperitoneal, the interstitial, and the submucous myomata. 1. The *subserous* or *subperitoneal myomata* (Fig. 5) are often pedunculate; they frequently cause changes in position and dislocations of the uterus. 2. The *inter-*

FIG. 5.—Intramural Myomata of the Uterus. These growths have begun their development in the body of the uterus. One of the larger ones— originally an intramural myoma—is now growing in the direction of the cavity of the uterus, and has already become submucous in character. Two others, growing in an outward direction, are already pushing up the peritoneal covering of the organ, and are making their existence appreciable from the outside as subserous myomata. The mucous membrane of the uterus is thickened.

stitial myomata (intraparietal or intramural myomata) may, as their growth progresses, become converted into the subserous or the submucous variety. 3. The *submucous myomata* are usually less bulky than either of the other varieties. They are not infrequently pedunculate and they resemble the ordinary polypi to a certain extent (they should be termed

myomatous polypi); but they are distinguishable from these by their firmer consistence and by the absence of cysts. They more or less completely fill the cavity of the uterus. Not infrequently dilated cyst-like cavities are disseminated through the substance of the myoma (myoma cavernosum cysticum). These owe their origin to the breaking down and liquefaction of the tissues at the corresponding spots (cysts due to processes of softening), or else they are formed from dilated lymph vessels and spaces in the tissues, and consequently represent genuine endothelium-lined lymph cysts. In these tumors there is a decided tendency (one-third of all cases) toward retrogressive changes—fatty degeneration, shrinking, and calcification, especially after the climacteric period has been passed. Interstitial calcified myomata (uterine calculi) may eventually lie free in the cavity of the uterus and be ejected during the patient's lifetime. Then, besides, a decided tendency to hemorrhages, inflammation, necrosis, and suppuration has been observed in these tumors, more particularly in the submucous myomata.

The *adeno-myomata* (of Von Recklinghausen) constitute a special group. They contain tubular gland structures, which come originally from the organ of Wolff; and often cysts, lined with cylindrical epithelium, are found in them (cystadenomata).

It is only in rare cases that myoma and carcinoma of the uterus occur together in the same case.

During the development of the myoma the uterus often undergoes at the same time a marked degree of hypertrophy, the cavity of the organ becoming dilated, and its walls—especially in the submucous

varieties—increasing in thickness up to the dimensions of a pregnant uterus. In the case of multiple intramural myomata the uterine walls are generally somewhat atrophied, owing to the fact that the larger part of the normal tissues of which they are composed has been destroyed by the advancing growth of the tumors.

Carcinoma of the Uterus.
(Plates 17 and 21.)

Cancer of the uterus is one of the commonest forms of carcinoma (one-third of all cases of cancer in women). It first manifests itself during the climacteric years or near that period of life.

The starting-point of the growth is most commonly the epithelium of the portio vaginalis of the uterus, less frequently that of the cervical mucous membrane, and the most rarely of all (between three and four per cent of all cases) the mucous membrane of the body of the uterus. The frequency with which cicatrices, inflammatory erosions, and other conditions of irritation are apt to be located in the neighborhood of the external orifice of the uterus explains why these parts show a local predisposition to the development of a carcinoma. The catarrhal affections of the cervix, which are of such frequent occurrence, are apt to lead to ectropium and to erosions.

Cancer of the Lower Part of the Uterus (carcinoma of the portio vaginalis and neck of the uterus).—The following varieties are distinguished: superficial or flat cancer, characterized by a tendency to spread along the surface; penetrating or infiltrating cancer, which very rapidly extends to the submucous and

muscular parts of the organ; and papillary cancer (cauliflower tumors), which, in the form of a cauliflower-like tumor of hemispherical shape, fills the vault of the vagina. These latter growths are generally very easily broken down, owing to their loose consistence.

The carcinomata which involve the neck and vaginal portion of the uterus begin as mere nodules or knobs, which rapidly break down and form ulcers; and these latter in turn present a finely nodulated surface with elevated margins, and they bleed easily. Another peculiarity of these cancers is shown in the fact that they tend to grow out laterally into the parametral tissues and also into the upper portions of the vaginal wall. In one-quarter of all cases a carcinoma of the vaginal portion eventually reaches the os internum of the uterus. The tendency to break down at an early stage and to form ichorous pus is a characteristic which belongs to all the different varieties.

Carcinoma of the body of the uterus seems to originate first in the normal uterine glands. It extends its growth as a rule along the surface, and is rarely characterized by the development of nodules. At first the mucous membrane appears to be thickened, the glandular parts showing an atypical proliferation of their epithelial structures; and then these proliferating masses of epithelium, after penetrating deep down into the layers of muscular tissue, cause these, through pressure, to melt away and disappear. Quite often papillary forms of cancerous growth are also encountered. When the irregularly shaped proliferating masses reveal the existence of tubules with a demonstrable lumen, it is customary to apply the

term *adeno-carcinoma* to these growths. In cancer
of the body of the uterus its internal surface will be
found to have been eaten away by deep ulceration,
and the uterine cavity will be correspondingly en-
larged. The disease also shows a tendency to involve
the neighboring peritoneum.

In the further growth of a uterine cancer it is a
common occurrence for the anterior wall of the vagina
to undergo complete destruction, while at the same
time the posterior wall of the bladder becomes infil-
trated with cancerous elements. In from twenty-five
to thirty per cent of these cases a vesico-vaginal fis-
tula will be formed. Furthermore, in a large majority
of the cases secondary carcinosis of the correspond-
ing lymph glands—especially the retroperitoneal,
prevertebral lymph glands, which lie at the entrance
to the pelvis—may be expected. Metastatic carcino-
sis of the liver, lungs, kidneys, and other organs
occurs more rarely. While the destructive process
is advancing toward the peritoneal lining of the
smaller pelvis, an inflammatory reaction is developed
in the latter, and as a result of this an obliterating
and adhesive pelveo-peritonitis follows, particularly
in the region of Douglas' cul-de-sac. It is only now
and then that the disease takes on the character of a
fatal purulent peritonitis. In about two-fifths of all
the cases the cancerous infiltration of the walls of the
bladder, in the neighborhood of the openings of the
ureters, leads to narrowing of these channels, and
this in turn induces hydronephrosis with a corre-
sponding obliteration of the renal substance.

Sarcoma of the Uterus.

Sarcoma of the uterus—either the round or the spindle cell variety—is, in comparison with carcinoma of this organ, observed only rarely. It occurs either in the form of a circumscribed growth or as a diffuse infiltration. It is usually located in the body or in the fundus of the uterus, more rarely in the cervical portion; in which location, according to recent accounts, endotheliomata have been seen. The starting-point of the growth is generally the mucous membrane or the submucosa, and only rarely the muscular layer and the subserosa. Occasionally a combination of sarcoma and myoma comes under observation.

ISEASES OF THE VAGINA AND VULVA.

Changes in the position of the vagina and vulva are sometimes seen, commonly in connection with dislocations of the uterus. These are prolapse of the anterior wall of the vagina (or *vaginal cystocele*) and prolapse of the posterior wall (or *vaginal rectocele*). In these conditions, as in the case of prolapse of the uterus, the mucous membrane of the vagina undergoes an epidermoidal thickening (pachydermia).

Inflammation of the vagina (colpitis, vaginitis) occurs both during the lying-in period and independently of this state.

Acute colpitis (vaginal catarrh) is often of gonorrhœal origin; it also may develop in consequence of some irritation of a mechanical, thermic, or chemical nature; and it sometimes results from the presence

of worms (oxyuris) which have found their way in
from the rectum. The mucous membrane, while pre-
senting the usual inflammatory alterations (such as
redness and swelling), will also be found to be cov-
ered with a mucous or a purulent secretion, and
often the urethral mucous membrane and that of the
cervix of the uterus present evidences of having par-
ticipated in the inflammation. In a few cases ab-
scesses of the glands of Bartholin constitute an ac-
companiment of the disease. During the lying-in
period the inflammation of the vagina often assumes,
as a result of the processes of a similar nature which
are going on in the endometrium, a diphtheroid char-
acter; that is, it produces an ichorous pus, and is
characterized by the casting off of veritable sloughs
from such spots as have been injured. Under these
circumstances there is danger of a secondary infec-
tious pericolpitis, a parametritis, a peritonitis, and
even of a general septic infection.

Chronic colpitis (chronic vaginal catarrh, fluor albus,
leucorrhœa) either grows out of some acute inflamma-
tory process, or else it develops in a primary manner,
in women who for any reason are in a weakened and
run-down condition. An excessive desquamation of
epithelial cells and the formation of exceedingly fine
villus-like granulations (colpitis granulosa) give to
the moderately reddened mucous membrane a cloudy
and velvety appearance. There is also such a thing
as a fibrous and adhesive colpitis. It is generally
associated with some injury or loss of substance, and
it is likely to result in narrowing or atresia of the
vagina.

Syphilitic ulcerations, broad condylomata, and

cicatrices are often encountered in the fossa navicularis; and as a secondary development there may appear, in connection with these, a chronic ulcerating proctitis and periproctitis, a process of disease which is likely to result in the production of callosities in the soft parts and in the formation of fistulæ (recto-vaginal fistulæ).

Injuries and ruptures of the vagina occur most often as a result of childbirth or of some violent mechanical interference (as with instruments). These tears or ruptures are generally located in the side walls of the vagina and they usually follow a longitudinal direction; they may also be situated in the vault of the vagina.

Tears at the entrance to the vagina and in the perineum are of frequent occurrence in connection with the act of giving birth to a child. Aside from simple superficial tears of the frenulum, four different varieties of ruptured perineum are distinguished, viz.: 1, *rupture of the perineum of the first grade*, in which both the frenulum and the vestibular portion of the perineal mucous membrane are torn; 2, *rupture of the perineum of the second grade*, in which the tear extends as far as to the sphincter ani; 3, *central rupture of the perineum*—a canal-shaped rupture, which extends from the vagina through the perineum, but does not involve the anterior part (the frenulum); 4, *rupture of the perineum of the third grade*, or complete rupture of the perineum, in which condition the perineum throughout its entire extent (including a part of the rectum) is torn. As a rule, injuries of this nature are likely to heal by the formation of a cicatrix. But then also, on the other hand, the torn sur-

faces may afford an entrance to septic germs, and in this way a general puerperal infection may take place.

As a result of the necrosis of certain areas of tissue which have been subjected to excessive pressure during the birth of the child, abnormal communications are often established between the vagina and neighboring organs (the bladder and the rectum, for example).   In this way are developed the pathological conditions known as vesico-vaginal fistula, recto-vaginal fistula, and vesico-recto-vaginal fistula.

New Growths.—*Carcinoma* is rarely seen as a primary growth in the vagina, and then usually in the form of a superficial cancer.   On the other hand, it often develops in this locality as a secondary manifestation; and in that case it will generally appear first in the upper and posterior portion of the vagina —that is, at the spot which a uterine cancer would naturally reach first in its downward growth.   Eventually there will be found in this region, in the place of the vault of the vagina and of the vaginal portion of the uterus, a veritable cavern whose walls, which are infiltrated with the cancerous growth, pour forth an ichorous pus.   In a few instances a primary cancer develops in the *labia*, the varieties observed being the papillary, the superficial, and the deep-seated cancers.   Benign papillary epitheliomata are found both at the entrance to the vagina and on the labia, and particularly often in connection with a preceding gonorrhœal colpitis and urethritis.   These growths are the so-called *pointed condylomata.*

In the tissues of the labia connective-tissue tumors (*soft fibromata*) sometimes develop.   At the start they appear to have simply an inflammatory character.

In their further growth they may attain almost any size—from that of a walnut to that of a child's head, or even a larger size (*elephantiasis vulvæ*). Tumors of this nature present a knobbed appearance, their surface being often lobulated or like that of a cauliflower.

Other Kinds of Tumors.—Myomata, sarcomata, and atheromata are rarely encountered, either in the vaginal walls or in the vulva. Cysts containing either a watery fluid or a mixture of mucus and blood are now and then seen in the walls of the vagina; and cysts containing gas (emphysema of the vagina, colpohyperplasia cystica) are seen even more rarely than these.

Soft and indurated ulcers of venereal origin are often met with upon the inner surface of the nymphæ and labia majora, on the commissure, and in the vestibule.

DISEASES OF THE FEMALE MAMMARY GLANDS.

Imperfect development of the mammary glands—*hypoplasia*—may be attributed to various causes. It is one of the manifestations of a general subnormal development of the entire body; it is often associated with defective sexual development (rudimentary state of the ovaries and uterus); it may also be due to wearing unsuitable clothing which exerts pressure upon the glands while their development is progressing; and, finally, it is extremely common in women who are in other respects normally developed, but who have inherited a predisposition to imperfect development of the breasts. The transmission of such a predisposition from parent to offspring is some-

thing which evidently must be connected with the prevailing practice of not nursing one's own child. When, through a long series of generations, either no demands at all or only slight ones are made upon these glands, they become defective in their development and functionally atrophied; and this condition may then be transmitted regularly from mother to child.* The striking predisposition which the mammary gland manifests to become the seat of all sorts of benign and malignant new growths must surely have some connection with this degenerative hypoplasia. And in this respect the gland shows some similarity to the imperfectly developed, functionally useless, non-descended testicle lodged in the inguinal canal, inasmuch as malignant new growths show a special predilection for this organ when placed under the conditions named.

Atrophy of the mammary gland is sometimes observed in general cachexia, and also in general obesity (in which latter condition an excessive development of fat and defective functional power characterize the "fleshy breasts"—the term which is commonly applied to them under these circumstances). A physiological atrophy takes place during the climacteric years, and then a compensatory development of fat (*ex vacuo*) makes good the loss due to the wasting away of the gland.

* In Germany it has been found that on the average only from one-half to two-thirds of all the women who give birth to a child are able to provide it with sufficient milk from their own breasts for a period of ten days (!) ; and only thirty per cent of the women can do this for a period of half a year.

INFLAMMATION OF THE MAMMARY GLAND; MASTITIS.

Except during the lying-in period inflammation of the mammary gland is very rarely seen, and then almost always as a result of some injury. It runs a mild course, and terminates either in resolution or in the formation of an abscess. When an abscess of this kind is deeply seated, it may easily be mistaken for a tumor.

By far the commonest form of inflammation is that which occurs during the lying-in period—*puerperal mastitis*, which usually is of infectious origin. About six per cent of all nursing mothers suffer from the disease, which develops as a rule during the month immediately following the birth of the child.

In a smaller number of cases the inflammation (under the influence of an infection by streptococci) confines itself to the surface and takes on an erysipelatous character. It starts from a fissure or other lesion of the nipple, and runs a course not unlike that of an erysipelas migrans or a phlegmon, the parenchyma of the gland participating in only a subordinate manner in the disease. Precisely as takes place in infection of a wound, the pathogenic germs gain an entrance from the erosions which so commonly are present in the nipple, and work their way into the lactiferous ducts. The coagulation and subsequent decomposition of the secretion of the gland are favoring factors, the action of these solidified matters being that of foreign bodies.

The most common form of inflammation is the *circumscribed parenchymatous and interstitial mastitis,*

in which only a segment of the gland is involved
(usually the lower and outer portions only). The
lobule which is inflamed presents a swollen appear-
ance, and the skin which covers it is reddened. In
the inflamed area an infiltration of cellular elements
takes place in the intralobular connective-tissue frame-
work, and the tissues break down into pus—in a word,
the inflammation results in the formation of an ab-
scess. This may empty itself by breaking through
into the lacteal ducts, in which case the pus can be
pressed out through the openings in the nipple; or
the rupture may take place outwardly through the
skin; or, finally, the collection of pus may be emp-
tied by means of an incision. When an abscess has
thus been laid open, its walls will be found to be fis-
sured and covered with shreds. There may be quite
a large number of separate abscesses, and in some
cases new ones are constantly forming and breaking
through. These fistulous tracks may also — when
they happen to communicate with lacteal ducts—dis-
charge milk. In this manner milk fistulæ are devel-
oped, and they may persist for a long time afterward.
In rare cases an enveloping capsule may form around
the abscess, and the contents of the latter may then
undergo, first, inspissation, and afterward calcifica-
tion. A purulent mastitis rarely terminates in a
severe general infection and septicæmia. In the ma-
jority of cases the inflammatory products are ab-
sorbed and the parts return to a healthy condition.
In some instances the recovery is incomplete; that
is, indurated areas (milk nodes) remain behind.
These, when more closely examined, are found to
be nodulated masses of hyperplastic connective tis-

sue which contain in their substance lacteal ducts, the majority of which are dilated.

Tuberculosis of the mammary gland, in the form of a tuberculous inflammation, is of very rare occurrence. On the other hand, mammary tuberculosis in different forms (miliary eruption, diffuse tuberculous inflammation) is often observed in the cow. (Hence the danger, for man, of an infection of the milk.)

NEW GROWTHS OF THE MAMMARY GLAND.

The *connective-tissue tumors* of the breast begin their first development in the interacinous, the interlobular, or the periglandular framework of the organ, and they often include in their substance glandular elements and cysts.

The lipoma, which originates in the fatty tissue surrounding the gland, is a growth of rare occurrence; and so too are the myxoma, the myoma, and the chondroma. On the other hand, the fibroma—which is generally seen in the form of a mixed tumor, in combination with an adenoma (fibro-adenoma)—is encountered more frequently.

Sarcoma of the mammary gland is a tumor of comparatively common occurrence. It may present itself in the form of a circumscribed, nodular tumor, which grows out from a few lobules of the gland, or else it may disseminate itself diffusely throughout the entire organ.

The softer varieties of sarcoma are composed of round or spindle cells, while the harder forms are represented by the fibro-sarcoma. The giant-cell sarcoma and the melano-sarcoma occur very rarely. In sections of these sarcomatous growths the cut sur-

face presents a glistening moist appearance and a uniform grayish-white color. Fatty degeneration may be seen in a few portions of the tumor, and now and then softening, necrosis, hemorrhage, and cystic formations may be encountered at different points in the mass.

Adeno-cysto-sarcoma (cysto-sarcoma phyllodes proliferum or intracanaliculare) should be considered as a special variety of sarcoma, in which the newly formed tumor masses, covered with epithelium, grow like polypi or like leaves into the dilated lacteal ducts, which are also lined with small pavement epithelium. In this manner a tumor is developed, in which there are numerous irregularly shaped, cleft-like spaces; and when this lobulated tumor is laid open by an incision the cut surface presents very much the same appearance as does that of a head of cabbage, in which there are numerous layers of leaves. This adeno-sarcoma, which has usually a hemispherical shape, sometimes grows quite rapidly, and may attain a considerable size. Its malignity is mainly of a local character, showing itself in a tendency to involve neighboring organs. Ulceration sets in only at an advanced stage of the growth. Metastases to lymph glands and internal organs have not been observed.

Epithelial Tumors of the Mammary Gland.

Adenoma of the mammary gland is rarely encountered as a diffuse tumor involving both breasts. It occurs more frequently in the form of a circumscribed nodular tumor. It is a very common experience to find it in combination with one of the varieties of

connective-tissue growths—that is, in the form of a fibro-adenoma, a myxo-adenoma, or an adeno-sarcoma. When there is a tendency to the formation of cysts, there will be developed a cystadenoma. The adenoma is often of small size, and its growth advances slowly. Often, too, it serves merely as the forerunner of a malignant adenoma (adeno-carcinoma) or of a genuine cancer.

Carcinoma of the Mammary Gland.

Cancer of the breast is the most frequent carcinomatous disease of the female sex, and it represents about four-fifths of all mammary tumors.

The following forms are recognized: the carcinoma simplex, the carcinoma medullare, the carcinoma fibrosum, and the carcinoma gelatinosum or colloid carcinoma.

The *carcinoma simplex* occurs in two principal forms: the glandular and the tubular. At first it is a small nodular growth which is immovably attached to the tissues immediately around it; it is not separated from its surroundings by any sharp limits. The nodule is, as a rule, intimately associated with the overlying skin, and this is particularly the case when it is located near the nipple. When the tumor is cut open with the knife, there is presented to the eye a grayish-white area (corresponding to the cut surface of the nodule) which has a lardaceous appearance, and presents scattered whitish-yellow spots. When the edge of the knife is drawn over this cut surface a somewhat thick, grayish-white juice is obtained which under the microscope will be found to consist principally of epithelial elements, some of them

doubtless showing evidences of fatty degeneration. When the stroma is only scantily developed, while at the same time the epithelial elements predominate, the tumor will present a marrow-like consistence (*carcinoma medullare*, medullary cancer). On the other hand, when the stroma is powerfully developed the cancer will be of the hard variety (*carcinoma fibrosum*, scirrhus, atrophying cancer). This last form of cancer (ten per cent of all cancers of the breast) creaks when cut with the knife, and very little juice can be obtained from the cut surface.

The *gelatinous* or *colloid cancer* (carcinoma gelatinosum) is rarely seen in the mammary gland.

Cancer of the breast shows a disposition to involve, at a very early stage, the overlying skin. The tissues are apt to break down at this point, and then there remains a crater-like ulceration. In scirrhus, on the other hand, there is a tendency to shrinking, as a result of which there is apt to be a cicatricial drawing in of the surface of the skin. Then, besides, the carcinoma is likely to involve the neighboring muscular tissue and the pleura, either by a continuous extension of the growth or in a disconnected manner—*i.e.*, with normal tissues lying between the separate areas of tumor growth. In a very short time a metastatic transfer of cancer elements to the glands of the axilla takes place. It is indeed only in rare cases that these glands escape.

Cancer of the breast occurs most frequently in women who are between forty-one and fifty-five years of age, and it is encountered comparatively seldom before the thirtieth and after the fifty-fifth year of life.

Cancer occurs more frequently in women who bear

children than in those who do not; and women who
have gone through an attack of mastitis are more
than ordinarily prone to the disease. After the tu-
mor has been removed by operative measures, a recur-
rence may be looked for in the majority of instances.
A definitive cure is obtained in only a moderate pro-
portion (somewhere between twenty and thirty per
cent) of the cases, and then only when the operation
has been performed at a very early date, and when
the axillary glands have been removed at the same
time. It should be stated, however, that even when
the operation proves to be unsuccessful it neverthe-
less prolongs the patient's life.

Nervous Apparatus.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE DURA MATER.

Inflammatory processes involving the dura mater (*pachymeningitis*) are either benign in character or of infectious origin. According as the inflammation is localized upon the inner or upon the outer surface of the dura, it is customary to speak of it as a pachymeningitis interna or a pachymeningitis externa.

A very common form of the disease is that which bears the name of *chronic adhesive and fibrous pachymeningitis*. In this form the dura will be found to be more or less firmly adherent to the inner table of the skull, thickened (through increase of the connective tissue), and opaque. In the higher degrees of the disease this membrane feels stiff and tough like leather. In its character of bone periosteum this membrane, when inflamed, often produces thickening and hyperostosis of the skull cap—pachymeningitis ossificans. This form of periostitis occurs especially in old age, as an accompaniment of circulatory disturbances, chronic alcoholism, psychoses, etc.

Pachymeningitis Interna Hæmorrhagica.

(Plate 31.)

The commonest form of inflammation of the dura mater is that which is known as *pachymeningitis interna hæmorrhagica* or *vasculosa*. At the commencement of the disease the internal surface of the dura, especially over the cerebral hemispheres, will be found covered with a delicate, veil-like coating of a bright red or a dark red color, and easily removable from the membrane. This pseudo-membrane, which is composed partly of fibrin and partly of newly formed and quickly vascularized connective tissue, increases in thickness, sometimes quite fast, at other times more slowly; and in it may be seen, either with the unaided eye or by means of the microscope, both diffuse and punctate extravasations of blood. When the disease lasts for a sufficient length of time this highly vascular deposit or pseudo-membrane assumes a tougher consistence and adheres more firmly to the dura mater. When frequently repeated hemorrhages (by diapedesis) take place into the delicate inflammatory membrane, and particularly into those layers of it which lie immediately next to the dura, there will be formed—in the later stages of the disease, and especially when it lasts for a considerable time—a veritable sac of blood. This mass of blood spreads out over the surface of the brain; the only thing which limits its advance being the primary inflammatory pseudo-membrane. Ultimately it may attain a thickness of several centimetres and an extent equal to that of a large saucer, or even larger. To this con-

dition the term *hæmatoma of the dura mater* is commonly applied. Through the retrograde changes which take place in the red blood corpuscles and the coloring matter of this escaped blood the organized membrane soon assumes a rusty hue; and by aid of the microscope it will be found that this is due to the presence of free granular and crystalline pigment (hæmatoidin). The feature which is the most characteristic of this proliferative (and also somewhat hemorrhagic) inflammation is the rapid and at the same time prolific development of blood-vessels; and by reason of the imperfect organization of these vessels, which are evidently subjected to unfavorable conditions (*e.g.*, inadequate provision for the return flow of blood), repeated hemorrhages are likely to occur.

Hemorrhagic and vascular pachymeningitis is often found accidentally (*i.e.*, not having been suspected during the patient's lifetime), at the post-mortem examination, to have been the immediate cause of death. Then, besides, it is encountered under the following circumstances: in persons—men more frequently than women, and especially those who have reached middle life or old age—who are suffering from some form of psychosis, from progressive paralysis, or from atrophy of the brain; in cachectic individuals; and especially often in those addicted to the excessive use of alcohol. The diminution in volume of the brain as a whole (from atrophy and anæmia) induces compensatory hyperæmia of the dura mater (through diminution of the intracranial pressure), and thus plainly, in many cases, favors the development of this peculiar inflammation of the dura mater, which in the main is only scantily supplied with blood-vessels.

PACHYMENINGITIS PURULENTA.

According to the localization of the disease upon the outer or upon the inner surface of the dura, it is customary to apply to it the name of *external purulent pachymeningitis* or that of *internal purulent pachymeningitis*. The external variety usually develops as a secondary disease, after some injury to the bony walls of the cranial cavity, and especially after an injury to the vault of the cranium, with infection of the wound. It may also develop through an extension of the inflammation from some point in the neighborhood; and it is particularly apt to follow an otitis media purulenta or a caries of the petrous portion of the temporal bone. While it is true that an external pachymeningitis (associated with a partial separation of the dura from the inner surface of the skull cap) is encountered, as a rule, only as a circumscribed disease, nevertheless such a purulent inflammation of the inner surface may extend until it involves large areas of the internal surface of the dura. If an infectious process happens to be located in the immediate neighborhood of the wall of one of the sinuses, it is an easy matter for a secondary septic endophlebitis, with thrombosis (secondary septic thrombo-phlebitis), to develop; and, as a matter of fact, this complication, in the case of the transverse sinus, at the point where it passes over the posterior aspect of the petrous portion of the temporal bone, is often observed.

Syphilitic inflammation of the dura mater—*pachymeningitis gummosa*—occurs with special frequency

upon the outer surface of the dura, at the base and toward the front part of the cranial cavity; and generally with this process is associated an analogous

FIG. 6.—Spindle-Cell Sarcoma of the Dura Mater. In this patient, a boy nineteen years old, there had been growing slowly, for a period of two years previous to the time when he came under observation, a moderately hard, elastic tumor, which projected symmetrically above his forehead and over both temples. This tumor, which is somewhat pointed in front and rises upward somewhat like a helmet, is covered with a tensely stretched skin and is immovably attached to the underlying bone. Operative removal of the growth, followed by death at the end of four days. At the post-mortem examination it was found that the tumor sprang from the outer surface of the dura mater, had grown diffusely through the overlying bone, and had pushed its way up under the skin in the manner shown in the accompanying cut. A portion of the tumor, about the size of a hen's egg, had grown downward into the cranial cavity, where it occupied a certain amount of space on both sides of the falx, and of course made an impression of corresponding depth upon the cerebral hemispheres. A laminated thrombus was found in the longitudinal sinus.

A more detailed description by Dr. Carl Bartholomae will be found in Langenbeck's *Archiv für klinische Chirurgie*, Bd. x., 1869, S. 389.

inflammation of the soft membranes. Yellowish-gray, gelatinous growths, composed chiefly of round

and spindle cells, develop upon the dura mater. At a later stage, these growths assume a more distinctly yellow color, and show a tendency to undergo retrograde changes, such as fatty and cheesy degeneration. The dura mater of the spinal cord may be diseased in a similar manner; but in the majority of cases of *chronic syphilitic meningo-myelitis* the chief pathological alterations are found, not in the dura but rather in the soft membranes.

New growths of the dura mater are of rather common occurrence. They are represented by the following varieties: osteoma, fibroma, fibro-sarcoma, sarcoma, and psammoma. As a rule, they grow slowly, and attain only a moderate size. Generally, they grow from the internal surface of the membrane toward the cranial cavity, and as they advance they destroy a corresponding portion of the cerebral cortex. In other cases—especially those of sarcoma and osteoid sarcoma—they break through the skull and grow in an outward direction. (Fig. 6.)

THROMBOSIS OF THE SINUSES OF THE BRAIN.

Thrombotic coagulations occur with special frequency in the large venous blood channels of the dura mater. As is the case with thrombi of other peripheral veins, those which develop in these channels are either harmless—as in *autochthonous marasmic thrombosis*—or infectious, as when a septic endophlebitis is the cause of the localized coagulation of blood. An *autochthonous sinus thrombosis* is oftenest found in the superior longitudinal sinus, from which point, in a centrifugal direction, the coagulating process ex-

tends into the communicating veinlets which belong to the pia mater covering the convexity of the cerebral hemispheres. This dangerous affection of the veins, associated as it is with closure of the lumen of the vessel, occurs most often in cachectic and anæmic individuals whose heart power is enfeebled. Certain anomalies of the blood (more particularly chlorosis) predispose decidedly to such thrombosis; and so, too, do certain chronic conditions of inanition which are observed in childhood.

Among the results which follow the process described above, especially when the sinus rectus and the vena magna of Galen are implicated at the same time, acute phenomena of stasis in the areas belonging to these veins deserve special mention. Such are, for example, well-marked venous hyperæmia, œdema due to stasis in the venous circulation, multiple hemorrhages, red softening, particularly in the neighborhood of the walls of the ventricles, and acute hydrocephalus internus. It is an easy matter at the post-mortem table to mistake such apoplexies, due to stasis, for an acute multiple hemorrhagic encephalitis; and this is particularly likely to happen if the primary thrombosis escape attention.

Septic thrombo-phlebitis (primary phlebitis and secondary thrombosis) takes place most commonly in the transverse sinus, and often extends from this point, in a centripetal direction, on into the jugular vein. As a secondary result embolic abscesses may form in the lungs (general septico-pyæmia), or a purulent leptomeningitis may be developed (in more than one-third of the cases). It is only in exceptional cases that recovery takes place (either with or without an operation).

INFLAMMATION OF THE SOFT MEMBRANES OF THE
BRAIN AND SPINAL CORD — LEPTOMENINGITIS;
ARACHNITIS.

The commonest form is that which is known as
chronic fibrous meningitis. It is characterized by a
milky clouding of the soft membranes and by the de-
velopment of an unusually large number of Pacchi-
onian bodies or granulations. As a rule the disease
develops slowly, either as a consequence of frequently
repeated congestive hyperæmias, or as the sequel to
some preceding inflammatory process, or again as a
secondary result of any one of a variety of brain dis-
eases (*e.g.*, atrophy).

ACUTE PURULENT MENINGITIS.

This disease is encountered in two principal forms:
1, as a diffuse inflammation extending over the con-
vex and basal portions of the brain (*convexity menin-
gitis*); and 2, as a purulent basilar meningitis, asso-
ciated, as a rule, with purulent inflammation of the
membranes of the spinal cord (*epidemic cerebro-spinal
meningitis*).

1. *Diffuse convexity meningitis.* This form of the
disease develops in connection with injuries of the
skull or as a result of disease in the neighborhood
(such as caries of the petrous portion of the temporal
bone). It may also owe its origin to disease located
in the nasal cavities or in the frontal sinuses, or to
disease (abscess) of the brain; and, finally, it may
develop through metastatic processes (by way of the

blood channels), in the course of a septicæmia or a
pyæmia, of an erysipelas, or of a pneumonia. The
exudation is sero-purulent or fibrino-purulent in char-
acter, and is usually found distributed throughout the
meshes of the pia mater, and more particularly be-
tween the convolutions and in the immediate neigh-
borhood of the blood-vessels, which present the ap-
pearance of having whitish-yellow or yellowish-green
margins. The cerebral cortex, which participates to
a greater or less degree in the inflammation, is in a
congested state, and cellular elements are scattered
throughout its substance. Occasionally capillary
apoplexies are found in the cortex, in addition to the
alterations just enumerated—a combination of lesions
which justifies the use of the term *meningo-encepha-
litis*. Recovery is a rare event; in the majority
of instances death follows in the course of a few
days.

It very rarely happens, in these forms of menin-
gitis, or even in purulent epidemic cerebro-spinal
meningitis, that the purulent inflammation extends
to the plexuses of veins and to the ventricular epen-
dyma (thus setting up an endomeningitis). When
this extension does take place the contents of the
ventricles will be found to be more or less cloudy
—rarely of a sero-purulent character — and of a
yellowish-green color. To this condition the term
pyocephalus internus is commonly given.

2. Purulent basilar meningitis, or *epidemic cerebro-
spinal meningitis*. In this form of meningitis the in-
fectious process localizes itself at the base of the
brain and along the spinal cord. As the cause of
this inflammation—which almost always terminates

in death, recovery being a very rare event—a specific microparasite has been discovered, viz., the meningo-coccus intracellularis.   (Plate 28.)

TUBERCULOUS BASILAR MENINGITIS.

In a comparatively small number of instances a tuberculous inflammation of the soft membranes of the brain develops, as a secondary process, from older solitary tubercles of the brain itself, or from a tuber-culous otitis media, or from a tuberculous caries of the spine.   In the great majority of cases, however, the tubercle bacilli are transported along the blood chan-nels from some older tuberculous deposit—already cheesy and in necrotic condition—which is situated in another part of the body; and these bacilli almost invariably become lodged at the base of the brain, and more particularly above the chiasma and the pons and in that vicinity, from which region the tuberculous disease spreads laterally into the fossæ Sylvii, as far as to the island of Reil.   The older tuberculous deposits, from which this infection of the brain proceeds, may be located in the apices of the lungs, in the lymph glands, in bony structures, in the joints, in the kidneys, or in the testicles.   It is only in rare instances that the bacilli are transported to the convex portion of the brain (two per cent) or to the membranes of the spinal cord (two per cent). When death has occurred during the early stages of the disease a scanty, sero-gelatinous, pale yellow or grayish exudation will be found, at the post-mortem examination, in the interstices of the loose tissues composing the soft membranes of the brain, and es-pecially along the course of the blood-vessels.   As

one watches it this exudation will be seen to ooze out slowly from the tissues; and farther on in the course of the disease (say in the second week) it will be likely to present the characters of a fibrino-purulent exudation, in which the cellular elements are more numerous.   Then there is another alteration which, in this more advanced stage of the disease, will be likely to attract our attention, viz., the presence, in the lymph sheaths of the finer arteries, of very small nodules—some of them scarcely visible, while others are as large as the head of a pin—which have an opaque centre.   It will also be observed at the same time that, in the vicinity of the fossæ of Sylvius, the cerebral cortex is more seriously involved, for in this region it is congested, swollen, and in many portions full of small miliary hemorrhagic foci or apoplexies, all these lesions indicating the existence of a meningo-encephalitis.   Through the spreading of the inflam-mation, by way of the cerebral fissure, over the plexus of veins and the ependyma of the ventricles, a serous exudation, rich in cellular elements, may be expected to take place into the cerebral ventricles (*inflamma-tory hydrocephalus internus*).   These ventricles under-go speedy enlargement—sometimes to three or four times their normal size, and the enlargement of these cavities is particularly marked in the case of the an-terior and posterior horns of the lateral ventricles, which may attain the size of a hen's egg, or an even larger size.   Numerous leucocytes will be found in the fluid contained in such abundance in the ventricles. The basal and central portions of the brain which bor-der upon the ventricles are often also highly œdema-tous, and sometimes softened to the consistence of

gruel. The parenchyma of the brain is everywhere œdematous and swollen, the surface convolutions being thoroughly flattened and no furrows remaining.

Tuberculous meningitis is a disease which occurs chiefly during early childhood and youth, but it does sometimes also occur, in the case of human beings, in middle age and the advanced period of life. Now and then (say in four or five per cent of all cases) the tuberculous meningitis is simply one of the manifestations of a general acute outbreak of miliary tuberculosis.

SYPHILITIC MENINGITIS.

In *syphilitic disease of the cerebral membranes* (meningitis gummosa) the latter, as a rule, become diffusely infiltrated and thickened. The process manifests a tendency to spread, and eventually all the membranes of the brain are converted by it into a thick and tough, whitish, skin-like mass, which shows, at different points, cheesy degeneration and cicatricial sclerosis of the tissues. At a later stage the disease may involve the bony structures of the skull and the brain itself. The favorite seat of a gummous meningitis is at the base of the brain. The blood-vessels—especially the arteries—manifest quite regularly the tissue alterations which characterize an obliterating peri-arteritis, a meso-arteritis, and an endarteritis of syphilitic origin. One of the secondary results of these alterations is atrophy of the cerebral cortex. In the early forms of cerebral syphilis—those which develop only a few months after infection has taken place—the only lesions found may be simply those of a syphilitic arteritis in the neighborhood of the base of the brain.

In the case of the spinal cord one occasionally has an opportunity of seeing a *postsyphilitic chronic* lepto-meningitis, with which a secondary involvement of the spinal cord is associated (*chronic meningo-mye-litis*). In these conditions the vascular apparatus is the part chiefly affected; the blood-vessels—particu-larly the arteries—presenting the appearance (when seen in transverse sections) of thickened and sclerosed rings, with narrowed or even obliterated lumina. Then, in addition, many of the smaller vessels show evidences of having undergone hyaline degeneration. The soft membranes of the brain are opaque, thick-ened, and infiltrated with numerous round cells. In addition to the disease of the blood-vessels, the spinal marrow becomes sclerosed (chronic myelitis), and disintegration of the nerve substance takes place; at the same time, however, the neuroglia remains un-changed. In many cases the disease extends to the dura mater, causing a *pachymeningitis spinalis hyper-trophicans*.

HYDROCEPHALUS.

(Plates 26 and 27.)

DROPSY OF THE VENTRICLES OF THE BRAIN.

(Hydrocephalus internus.)

DROPSY OF THE SUBARACHNOID SPACES.

(Hydrocephalus externus.)

The accumulation of a watery fluid in the cavities of the skull is an event of common occurrence.

The majority of cases which belong in this category —cases that have generally developed slowly in adults

—should be classified as genuine cases of dropsy, inasmuch as they owe their origin to disturbances of the circulation (beginning either in the heart or in the lungs) and particularly to chronic stasis in the venous circulation. The pathological alterations observed are the following: After the removal of the dura, it will be seen that the subarachnoid spaces are well filled with a serous fluid, and that the ventricles of the brain, which are more or less dilated, also contain more than the normal quantity of serum. The soft membranes are generally somewhat thickened, and they show a milky opacity. Chronic hydrocephalus internus (without hydrocephalus externus) often develops in connection with such tumors of the brain (more particularly of the cerebellum) as are located in the vicinity of the cerebral fissure or of the walls of the ventricles.

A special form of chronic hydrocephalus internus et externus develops, as a secondary disease, in connection with atrophy of the brain; this is the form known as *hydrops ex vacuo.* One can readily understand that any diminution in the volume of the brain must necessarily, as a result of the unyielding character of the bony skull, be compensated for by an increase in the quantity of the cerebro-spinal fluid. Cases of this character are observed in connection with senile and presenile (particularly arterio-sclerotic) atrophy of the brain.

On the other hand, the majority of cases of hydrocephalus internus in children (hydrocephalus congenitus et acquisitus), as well as the hydrocephalus which is associated with basilar meningitis, belong in the category of serous inflammations.

Inflammatory conditions of irritation affecting the ependyma (chronic ependymitis, endomeningitis) and the plexus of veins, play an important part in these forms of hydrocephalus, the ependyma often being found in a thickened condition, and showing on its surface a network design and granulations like particles of sand.

In *congenital hydrocephalus internus* (Plates 26 and 27) an abundant quantity of clear serum is commonly found in the dilated, almost oval cerebral ventricles. The atrophic and imperfectly developed brain substance surrounds these dilated cavities in much the same way as would a mantle. The optic thalami and the corpora striata are spread apart and flattened. The bony walls of the skull show corresponding changes. The cranium, for example, presents a spherical form, like that of a distended bladder; the individual bones of which it is composed are noticeably thinned, and their edges are separated from one another by a broad space which is spanned by a membrane; the cranial sutures do not interlock, and the fontanelles are broadly open. It is often impossible to ascertain the cause of the disease; we simply know that syphilis in the parents confers a certain degree of predisposition upon the offspring.

In many cases the cause is doubtless to be sought for in some disturbance in the development of the brain. When the predisposition at the time of birth is slight, and then during the first years of life it increases to such a degree as to induce a fatal termination, we may speak of the case as one in which there is a combination of congenital and acquired hydrocephalus. Now and then a case is seen in which

the disease advances to a certain point and then stops.

In many cases the pouring out of fluid into the ventricles of the brain begins only after birth (acquired hydrocephalus). This is apt to take place in connection with rachitis.

DISEASES OF THE BRAIN.

DISTURBANCES OF THE CEREBRAL CIRCULATION.

The amount of blood contained in the brain is subject to marked variations. The color of the organ, as observed at the post-mortem examination, aids us, to a certain extent, in passing judgment upon this point. Thus, for example, the paler the brain is, the smaller will be the amount of blood which it contains; while, on the other hand, a rosy-red color and a dark grayish-red indicate the presence of an increased amount of blood in the organ.

Hyperæmia of the Brain.

In this condition the brain is enlarged, the convolutions are flattened, and the furrows are obliterated. When the organ is laid open with the knife the cut surface of the gray matter presents a dark grayish-red hue and numerous bloody points, which can be made to disappear when the edge of the knife is passed over the surface. Arterial hyperæmia is generally seen in connection with hyperæmia of the cerebral membranes, and its significance is merely that of a functional disturbance. On the other hand, in sunstroke it indicates the existence of inflammatory

action. A venous hyperæmia is observed when the return of the venous blood is hindered in some manner (as in death which originates at the heart, and in thrombosis of the large venous trunks).

Anæmia of the Brain.

In this condition the tissues of the brain have a pale hue, and when there is an entire absence of blood the white substance has a waxy white color. Very few bloody points are visible upon the surface of a section, or they may be altogether absent. The consistence of the brain is perhaps somewhat increased; but if œdema is present at the same time, it will be found somewhat diminished. Generally speaking, the diminished amount of blood in the brain is simply one of various phenomena indicative of a general acute or chronic anæmia (such as death from loss of blood, or some cachectic disease like tuberculosis, cancer, etc.). In other cases still, some local intracranial cause for the anæmia may be discovered; as, for example, a condition of hydrocephalus, a tumor, or a well-marked arterio-sclerosis.

Œdema of the Brain.

When the brain substance is abnormally saturated with serum it will be found to have an increased volume, and its convolutions will appear flattened out. The consistence of the organ will also be found to be more or less diminished—in extreme cases, up to the point of actual softening (white softening); this latter condition being observed for example, in the vicinity of the ventricles in hydrocephalus internus and in meningitis.

CEREBRAL HEMORRHAGE; APOPLEXY.

(Plates 32 and 33.)

Hemorrhages into the parenchyma of the brain occur from various causes:

1. As the result of a general hemorrhagic diathesis, such as is observed in certain severe infectious diseases, in scurvy, in pernicious anæmia, and in leukæmia, in which conditions the hemorrhagic foci are multiple in character and vary in size.

2. As secondary phenomena in connection with certain new growths (sarcoma, glioma), or with centres of softening which have resulted from processes of degeneration and necrosis.

3. As the result of some mechanical injury. In this case the apoplexies are usually located in the more superficial parts of the brain substance, or else in or near the walls of the cerebral ventricles. These apoplexies occur most frequently in consequence of injuries to the skull cap, such as are produced by a blunt force, by a ball shot from a gun, by a stab or other punctured wound, etc. They may also occur without any demonstrable injury of the skull cap, as in the so-called concussion of the brain, in which, besides a diffuse molecular injury of the cerebral cortex, capillary apoplexies, disseminated contusions, and centres of softening may be present; these lesions being of such a nature that they are very likely to lead to alterations in the blood-vessels and to *late apoplectic attacks*. In cases of contusions and mechanical injuries of the cerebral cortex the ganglion cells may undergo necrosis and calcification. A trau-

matic apoplexy is often associated with a meningeal hemorrhage upon the surface of the brain, and the location of these two lesions usually corresponds either with the spot at which the trauma first took effect, or with one situated directly opposite to it (*contrecoup*).   The course and terminations of a traumatic apoplexy are generally the same as those of the spontaneous variety.   In the newborn child traumatic hemorrhages of the cerebral membranes and of the brain itself are particularly liable to occur when the obstacles to the delivery of the child are of such a nature as to require instrumental interference.

4. As the result, more particularly, of degenerative disease of the arteries, in combination with hypertrophy of the heart.   Such spontaneous apoplexies in the substance of the brain occur chiefly at an advanced period of life; seventy-one per cent occurring beyond the age of fifty, seventeen per cent between forty and fifty, and only eight per cent between thirty and forty.

The spontaneous form of cerebral apoplexy (Plate 32) occurs most often in the region of the caudex cerebri (the corpus striatum, the thalamus opticus, and the nucleus caudatus), which receives its supply of blood from a branch of the arteria pro fossa Sylvii (arteria lenticulo-striata).   The hemorrhagic foci vary in size, the majority of them being as large as a plum or even as a goose's egg.   Very often the blood breaks through into the ventricles, which, under these circumstances, will be found filled with bloody serum and masses of blackish-brown clotted blood.   In addition to the accumulated blood there will be found, in these apoplexies, remnants of broken-down brain

substance; and, for a certain distance, in the neighborhood of such foci, the brain will be found to present an œdematous and swollen appearance, and a bright yellowish color, while the consistence of these portions will be found to be diminished.   When death does not immediately follow such an apoplectic stroke, a variety of changes may be expected to take place in the affected brain area.   In the first place, the focus diminishes in size as a result of the gradual absorption of both the fluid and the solid portions of the apoplexy.   When the latter have been converted into a fatty, emulsion-like fluid, numerous granule cells (phagocytes laden with fat) make their appearance. The coloring-matter of the blood also undergoes certain changes, and the red blood corpuscles become swollen, lose their color, and shrink.   From the sixth day onward, owing to the instability of the iron in the hæmoglobin, diffuse hæmosiderin will be found to be present, and this in turn speedily becomes converted into granules.   In the course of the third week free pigment appears for the first time.   After the splitting off of the iron, hæmotoidin will be developed (in the course of the seventh week); and this will be found in a free state, both in crystalline form and as amorphous masses.   The apoplectic cicatrix, which has developed in the manner described, presents an ochre-yellow or a rusty color.   If the apoplectic focus occupies a position at the surface of the brain, a scar-like defect will remain as a permanent lesion.   On the other hand, if the focus be situated in the interior of the brain, a cyst with rust-colored walls will be the ultimate result of the apoplexy.

As regards the causes of spontaneous cerebral apo-

plexy, it has been shown that in many cases—aside from the influence of a certain degree of inherited predisposition—it is mainly due to two factors, viz.: *disease of the finer arteries* (with or without the formation of miliary aneurisms) and the *persisting increase in arterial pressure which is associated with hypertrophy of the left ventricle of the heart.* The plethora resulting from this increased arterial pressure also exerts a favoring influence. Among the diseases of the finer arteries may be mentioned fatty and hyaline degeneration, atheromatosis, and syphilitic endarteritis. In many cases—particularly those in which no miliary aneurisms exist—it may be assumed that a previous degeneration of the brain substance first brings on a sort of arterio-sclerotic, prehemorrhagic softening, and that this in turn produces a local predisposition to the occurrence of an apoplectic attack. Previous injuries (concussion, for example) may also induce—particularly in the walls of the cerebral ventricles—some local pathological change or degeneration which favors a *late apoplectic attack.* This is particularly true in the case of children and young individuals. It is a very common experience to find a fatal apoplexy in a case of chronic nephritis (Bright's disease), which, as is well known, is generally associated with hypertrophy of the left ventricle of the heart. This renal form of apoplexy is encountered particularly often in men who are between thirty and sixty years of age. Inasmuch as a cardiac hypertrophy which is not of renal origin predisposes in a much less marked manner to apoplexy, we may assume that the kidney disease—possibly through the intervention of the resulting hydræ-

mia—exerts a specially unfavorable influence upon the cerebral arteries. Whereas in advanced life arterio-sclerosis – generally in combination with hypertrophy of the heart—plays the leading part in the pathogenesis of spontaneous cerebral apoplexy, in younger persons (from twenty-five to fifty years of age) the main influences in bringing on a hemorrhage of the brain are, in addition to nephritis, syphilis, alcoholism, certain infectious diseases (typhoid fever, small-pox, pyæmia, tuberculosis), and, finally, abnormities in the blood-making powers (such as pernicious anæmia, scurvy, leukæmia).

SOFTENING OF THE BRAIN; ENCEPHALO-MALACIA.

This pathological change, in the great majority of cases, is of vascular origin; it develops in connection with thrombosis and embolism of the arteries, and with obliterating endarteritis (arterio-sclerosis).

The so-called *white softening* is most often encountered, in the form of separate foci, in that part of the brain which is supplied by the arteria pro fossa Sylvii (oftener on the left side than on the right), after embolism of this artery has taken place, particularly in the vicinity of the island of Reil and near the capsula interna. The emboli emanate from thrombotic and inflammatory processes involving the left side of the heart, the ascending aorta, and, more rarely, the pulmonary veins (those portions which are near the point where they enter the left auricle). In that portion of the brain which is suddenly cut off from its arterial blood supply, the following series of changes takes place: ischæmic necrosis; rapid break-

ing down, softening, and melting of the tissue ele-
ments; and the accumulation, in the area involved, of
wandering cells, which, by aid of the process known
as phagocytosis, have filled themselves full of a finely
granular fat—the principal product resulting from
the disintegration of the necrotic brain substance.
(Cells in this condition are known as granule cells.)
The brain substance, which at first was simply œdem-
atously infiltrated, will now be found in a half-soft-
ened state, almost fluctuating. This softened portion,
which may reach the size of a hen's or a goose's egg,
has no sharply defined limits; and very soon it is
quite sure to melt down into a fluid resembling pap
or imperfectly coagulated milk. Under favorable cir-
cumstances, if death do not in the mean time put an
end to the disease, the fatty and necrotic products of
the disintegration of the brain substance undergo
complete absorption and there remains only a cyst,
which is filled with a serous fluid, and whose walls
are lined with a delicate membrane of connective tis-
sue. Several such old apoplectic foci and cicatrices
are often found in the same brain—a fact which is
easily explained when we remember that such proc-
esses as embolism, thrombosis, and obliterating
endarteritis are constantly recurring; and these, of
course, must create, over and over again, some new
local disturbance.

INFLAMMATION OF THE BRAIN; ABSCESS.

Abscesses of the brain are, on the whole, of rare
occurrence. They may develop in consequence of an
injury of the skull or through an extension of the dis-

ease from a purulent and infectious process located in the neighboring bone (*e.g.*, caries of the petrous portion of the temporal bone, and septic thrombo-phlebitis of one of the sinuses). These latter abscesses, which are known as otitic brain abscesses, are located either in the temporal lobe or in the cerebellum. Brain abscesses may also develop by metastasis from disease of the lungs (putrid bronchitis, in cases of bronchiectatic cavities, empyema), or from a septic endocarditis of the left heart, in which disease multiple hemorrhagic foci also develop in the brain. Brain abscesses which originate in these ways may attain the size of a goose's egg, and not infrequently—particularly in septico-pyæmia—several of them may be present in the brain at the same time. In abscesses which have persisted for some time a sort of capsule develops around the collection of pus. If the abscess is situated near the surface of the brain, it may give rise to a secondary purulent leptomeningitis, the issue of which is likely to be fatal. In rare cases the pus of such an abscess has a foul odor and an abnormal color—circumstances which are due to the penetration of bacteria of decomposition into the substance of the brain (putrid abscess). Through the seemingly purulent softening of cheesy solitary tubercles a tuberculous brain abscess may develop.

A purulent and gangrenous inflammation of the brain is sometimes observed after a severe injury, which has caused a comminuted fracture of the skull and has laid bare the brain. It is also seen in cases of malignant new growths, which destroy the bony structures of the head and the membranes of the brain, and expose to view the cerebral cavity. (Plate 49.)

An encephalitis, which generally occurs in the form of separate hemorrhagic foci (*hemorrhagic encephalitis*), is observed now and then in the course of certain infectious diseases, such as influenza, typhoid fever, scarlet fever, and measles. In autochthonous marasmic thrombosis of the sinuses of the dura mater, and also of the veins belonging to the soft membranes of the brain (*e.g.*, the vena magna of Galen), multiple hemorrhagic foci are also observed. These, it must be remembered, may at times be mistaken for a hemorrhagic encephalitis.

As a cause of *cerebral infantile paralysis* we sometimes encounter an acute encephalitis, either congenital in its nature or acquired during early childhood. The disease leaves behind such demonstrable lesions as cicatrices and losses of tissue (porencephalia).

ATROPHY OF THE BRAIN.

Simple uncomplicated atrophy of the brain is observed most commonly as a senile or presenile affection. The brain—that is, the cerebrum—is more or less diminished in weight and volume; the cerebrospinal fluid, both in the subarachnoid spaces and in the dilated cerebral ventricles, is increased in quantity (hydrocephalus *ex vacuo*); and the soft membranes of the brain are opaque, as if infiltrated with milk. The convolutions are strikingly narrow, almost pointed, and the furrows are noticeably broadened. The organ as a whole is firm and tough, almost like leather. When it is cut open, it is easy to see, on the surface of the section, that the gray matter of the cortex is markedly diminished in breadth, and that

the white substance has also undergone some diminu-
tion. Oftentimes the skull cap will be found to be in
a thickened state. The larger and medium-sized
arteries are generally in a sclerosed condition.

The presenile form of diffuse brain atrophy, which
occurs in men between the ages of forty-five and sixty,
presents very much the same anatomical changes as
does the senile form; a highly developed atheroma-
tosis, especially of the basilar arteries, being demon-
strable in most cases as the chief cause of the disease.
The brain, like the heart, is much more capable of
withstanding the effects of inanition, of chronic anæ-
mia, than are the other organs of the body.

Diffuse atrophy of the brain is also observed in
cases of chronic hydrocephalus (Plate 26) and of
paralysis. In the latter condition the cerebrum
often loses more than one-quarter of its weight, while
the cerebellum remains almost unchanged. In a
large proportion of the cases the underlying cause is
a postsyphilitic process, associated with atrophy of
the nerve fibres and cells of the cerebral cortex; and,
as secondary results of these changes, we have nar-
rowing of the cortex and diffuse atrophy of the brain.

Local atrophy, in the form of separate foci or areas,
is generally the result of pressure, such as may be
exerted by tumors of the brain, by infectious granulo-
mata, by exostoses, and by parasites.

Defective development of the brain (hypoplasia),
or *microcephalia*, which is due to some arrest in the
general development, affects the organ either uni-
formly, as a whole, or else only in certain of its parts
—as, for example, the corpus callosum, the cerebel-
lum, or a single hemisphere. Children born with

this condition often grow up as dwarfs; they are simple minded or idiotic, and they generally have some other troubles, such as pareses, or anæsthesiæ, or contractures. The brain, in these cases, may weigh as little as three hundred grams, or even less. Microcephalia is either a congenital condition, depending upon harmful influences which have made themselves felt at the time of conception or during intra-uterine life, or else it is one which is acquired after birth. In the latter event the children are born in a normal condition, but, as a result of various local or general pathological processes, which cause circulatory disorders and different kinds of inflammations, the development of their brains is affected injuriously. Finally, the premature synostosis of the bones which form the base of the skull, as well as the premature union of the sutures of the skull, is also competent to cause an arrest in the development of the brain and to lead to hypoplasia of this organ—to microcephalia.

PORENCEPHALIA.
(Plates 34 and 35.)

Defects and fissure formations in the cerebral cortex are designated by the term porencephalia. These defective spots are lined with pia, filled with serum, and covered over with the arachnoid. In a certain proportion of the cases the condition is to be considered as one of *arrested development* (local aplasia), and is to be attributed to a cessation of growth, during fœtal life, in certain portions of the cerebral mantle. Then, through the overgrowth of neighboring parts of the brain, and through the accidental devel-

opment of hydrocephalus, a variety of forms of this condition is produced. Porencephalia sometimes furnishes the anatomical basis of cerebral infantile paralysis. A porencephalia often extends into the ventricles; and when this is brought about by the destruction of the corresponding portions of the cerebral cortex during intra-uterine life, or in early infancy, through some pathological process (such as hemorrhages, inflammations, etc.), it is proper to speak of it as a *secondary porencephalia*. Apoplectic cysts, such as that shown in Plate 35, may also develop after the period of childhood, through a mechanical injury or an inflammation, or by reason of some disturbance in the circulation (leading to softening). A dropsy of the ventricle favors the establishment of a communication between the defect and the ventricle, and it also exerts a determining influence upon the shape of the defect.

Cystic degeneration (intracerebral and subarachnoid cysts) is often observed in the brain of paralytics, and is to be attributed to obstructed circulation of the lymph and to the occurrence of an adhesive arachnitis in the fissures between the convolutions. Such cysts are often arranged like a string of pearls.

TUBERCULOSIS OF THE BRAIN.

(Plate 36.)

Tuberculosis of the parenchyma of the brain develops almost exclusively in the form of the so-called solitary tubercles or conglomerated tubercles, and much oftener (seven times) in children than in

adults.* In most instances the subchronic or chronic solitary tubercles owe their origin in the brain to a metastatic process; they rarely develop primarily in that organ. Among the tumor-like affections of the brain the solitary tubercle occupies the most important place.

The first beginnings of a tubercle are to be found in those vascular processes of the pia which extend into the peripheral portions of the brain. The nodules and nodes which originate in this way grow slowly, are usually of a rounded form, and may attain the size of a cherry, or an English walnut, or even a larger size. The nodule is mainly of a yellow color, cheesy in its composition, fairly tough as regards its consistence, but sometimes also soft, as when it is breaking down into pus. The marginal zone is of a grayish-red color or of a transparent gray, and full of miliary nodules which become blended with the larger parent nodule. The latter may therefore be considered as a conglomeration of what were originally much smaller nodules. The brain substance in the neighborhood of the solitary tubercles is often in a condition of red or white softening, as shown by the presence of capillary apoplexies in these parts. The solitary tubercles are most frequently located in the gray substance, particularly in that of the cerebellum, and also in the cerebral cortex, in the pons, and in the ganglia of the caudex cerebri. In from twenty to twenty-five per cent of all cases tuberculous granulo-

* Among one hundred and fifty cases of fatal tuberculosis in children (under fifteen years of age) the soft membranes were involved forty times, whereas the brain itself was affected only twelve times.

mata make their appearance in varying numbers. A fatal termination, through secondary tuberculous leptomeningitis, is a frequent occurrence. Secondary hydrocephalus internus is also a common complication.

TUBERCULOSIS OF THE SPINAL CORD AND ITS MEMBRANES.

(Plate 29.)

This disease is encountered in the form of a meningo-myelitis, with an eruption of miliary tubercles. Or, as happens in the brain, although much more rarely, solitary tubercles—either with or without secondary tuberculous meningitis—develop in the spinal cord. (More than one such tubercle is not often seen.) Both clinically and anatomically the results of such long-persisting and slowly growing nodules of conglomerated tubercles are very much the same as those which are observed in cases of tumor of the spinal cord.

Spinal pachymeningitis externa tuberculosa (Plate 29) is quite often associated with tuberculous spondylitis; the tuberculous process, under these circumstances, extending by direct continuity from the original seat of the disease to the external surface of the dura mater, and there setting up a circumscribed inflammation accompanied in part by proliferative, in part by necrotic phenomena. In this form of disease the neighboring spinal cord is subjected to changing conditions of pressure, and these give rise to what is known as compression myelitis.

SYPHILIS OF THE BRAIN.

Syphilomata of the brain (gummata) belong among the late products of syphilis. They are more frequent in adults than in children and youthful individuals. These growths vary in size, and there are often several of them in the same brain. In their early stages of development they are of a soft, gelatinous consistence. They are at times more or less intimately associated in their development with a gummous meningitis; then, again, at other times, they develop independently in the brain substance. In the more advanced stages of their growth the character of these syphilomata changes, one part becoming tougher in consistence, while another undergoes partial cheesy necrosis. While they are in this condition it is an easy matter to mistake syphilomata for solitary tubercles. Syphilitic arteritis is often present in the brain at the same time. Then, besides, still other pathological alterations of a syphilitic character are generally found simultaneously, not only in the brain (*e.g.*, multiple centres of softening in the cerebral hemispheres), but also in other parts of the body.

TUMORS OF THE BRAIN AND SPINAL CORD, AND OF THEIR MEMBRANES.

Malignant tumors (carcinoma, sarcoma) may develop in the brain, through metastatic agencies.

The following varieties of tumors are observed as *primary growths* in the brain: Glioma, glio-sarcoma, and all the different varieties of sarcoma (glio-sar-

coma, angio-sarcoma, myxo-sarcoma, fibro-sarcoma, and endothelioma), beside the psammoma, the cholesteatoma, and the dermoid growths of the meninges; which latter tumors develop from dislodged germinal elements, or they are endotheliomata (meningeal pearl tumors) which have developed originally from the endothelia of the cerebral membranes.

Generally these different tumors occur only singly in the brain. In rare instances, however, more than one may be found. They vary in size from the dimensions of an English walnut to those of a hen's egg, or even of a man's fist. They are found most often on the surface of the brain, in the neighborhood of the walls of the ventricles, and in the cerebellum. Their predilection for these particular parts of the brain is closely connected with their origin in some mechanical injury—an origin which can often be demonstrated. The gliomata and sarcomata are generally of a semisolid consistence (fairly fluctuating under digital pressure). They have a grayish color and a moist, glistening surface; and in some forms— particularly the angio-sarcomata—hemorrhagic foci are disseminated throughout the substance of the tumor. (This last fact explains the employment, in earlier days, of such terms as hemorrhagic sarcomata, fungus hæmatodes, and *Blutschwamm*.) When the tumor grows to be of considerable size, and when it has been in existence for a long time, some portions of it (especially the central portions) present an opaque and yellowish appearance, indicative of the necrosis and fatty degeneration that have already developed at these points in consequence of defective nutrition of the tissues.

The *psammomata* which originate in the plexuses of veins and the soft membranes of the brain are in reality fibro-sarcomata, *i.e.*, slowly growing tumors, in which are embedded concentrically laminated chalky bodies that have both the outward likeness and the physical properties of brain sand, as it is encountered in the normal plexuses of veins and in the pineal gland.　Tumors of the brain lead to wearing away and destruction of brain substance, to circulatory disturbances, to hydrocephalus internus, and to an increase of the intracranial pressure.

Tumors of the spinal cord and of its membranes come only rarely under observation, either as metastatic (in the case of carcinoma and sarcoma) or as primary growths.　The latter, so far as their general features and structure are concerned, are similar to the tumors of the brain.　The varieties most often encountered are the glioma, the sarcoma, and the glio-sarcoma.　The location of these tumors is either in the substance of the spinal cord (intramedullary growths) or in its membranous envelopes (meningeal tumors, either intradural or extradural in their location).　Such tumors either grow around the cord like a circular band, or they have a spherical shape; or, finally, the pia is infiltrated throughout its entire length by the tumor elements, so that the growth envelops the cord like a mantle.　Tumors of this sort rarely spread from the pia to the spinal cord.　When they grow beyond the size of a cherry or a hazelnut, they are likely to cause grave disturbances, such as atrophy of the cord due to compression, or its conversion into a flat band, or, finally, the so-called compression myelitis, with softening of the substance of the

cord at the affected points and an extension of this destructive metamorphosis both upward and downward.

The *parasites* which are occasionally found in the membranes of the brain and spinal cord are the following: the cysticercus cellulosæ, either only a few scattered individuals or large numbers of them; the echinococcus—very rarely; and, even more rarely, the actinomyces fungous growth, which may reach these parts either by aid of a metastatic process or by the route of continuous advancement. The latter parasitic growth appears very rarely, as a primary manifestation, in the ventricles of the brain; and, when it does so, it gives rise to the development of a myxomatous tumor.

HEMORRHAGES IN AND INJURIES OF THE SPINAL CORD.

The spontaneous escape of blood into the substance of the spinal cord—in a manner analogous to what takes place in cerebral apoplexy—is something which practically never occurs. The arrangement and situation of the arteries in this region serve to protect them against the high pressure and the resulting alterations (degenerations, dilatation, and rupture) which, in the case of the brain, constitute the anatomical groundwork of a spontaneous hemorrhage. Capillary apoplexies in the spinal cord, with secondary softening, are observed in the course of certain forms of poisoning (phosphorus, arsenic). They are also observed in cases of embolic plugging of the smallest blood-vessels; and here, too, the apoplexies are situated in the gray matter of the cord, in the vicinity of the central canal.

The hemorrhages which occur in the spinal cord are generally of traumatic origin, and are encountered with special frequency in the gray matter; within which the escaping blood, as it spreads, assumes the shape of a tube. This condition has been termed a *central hæmato-myelia*. Hemorrhages of this nature may take place even as early as during the birth of the child, and may thus give rise, at some later date, to a *syringomyelia*.

Among the traumatic affections of the spinal cord *concussion* of this organ plays an important part. Out of such a concussion a variety of anomalous conditions may develop. Thus, for example, it may induce a simple necrosis of one portion of the nervous structures of the cord; the breaking down of these elements taking place gradually, and the interstitial tissue either remaining unaffected or else manifesting a secondary proliferative tendency. In a second group of cases both the nervous elements and the supporting framework may undergo necrosis at the same time (a lesion of the cord in transverse section, with diffuse softening), while later there will be secondary degenerations and cavities will form. In a third group, circumscribed foci of necrosis establish themselves in the cord—foci which are similar in character to the embolic centres of softening in the brain, and which end in forming simple cavities. Finally, in a fourth group the two pathological processes of gliosis and cavity formation (syringomyelia) are brought into activity, in very much the same way as happens in the formation of a tumor through traumatic influences. The development of cavities is brought about by the destruction of neuroglia. In punctured wounds

of the spinal cord an œdematous swelling of the adjacent parts invariably follows, and in this way a certain number of nerve fibres are for a short time functionally disabled. After the swelling has subsided the affected nerve paths, or at least a part of them, resume their functional activity.

Traumatic influences are also responsible for those *meningeal hemorrhages* which are localized either in or between the membranes (intrameningeal apoplexies), and which, like a mantle, envelop the spinal cord. Such extramedullary hemorrhages may also take their rise from some point at the base of the brain— as a result, for example, of a spontaneous rupture of an aneurism situated in this locality. A hemorrhage like this is termed a descending spinal meningeal apoplexy.

COMPRESSION MYELITIS.

(Plate 37.)

Degenerative Atrophy of the Spinal Cord due to Compression.

Compression of the spinal cord owes its origin, in the majority of instances, to some disease of the spinal column (such as tuberculous spondylitis, with caries and necrosis; fractures and luxations, and new growths, of the bones) or of its membranes (inflammatory processes of a suppurative, syphilitic, or tuberculous nature; new growths); or it may be brought about by the development of a new growth in the substance of the spinal cord itself. As a general rule the spinal cord is able to bear, without any perceptible bad effects, a very considerable degree of

narrowing of the vertebral canal; but then again, at
other times, even a moderate degree of pressure seems
to suffice to destroy some of the nerve fibres, and thus
to effect a partial interruption of the communications
which pass through the cord. A compressing force
which is kept up for some length of time causes a
flattening of the cord and induces disturbances of
nutrition; and, as a secondary result, it leads to de-
generative atrophy of the fibres and nerve cells, and
yet without causing any alterations to take place
which might be termed inflammatory in their nature.

POLIOMYELITIS ANTERIOR ACUTA.
(Spinal Infantile Paralysis.)

This disease is located in the gray matter of the
anterior horns. It is probably of vascular origin and
dependent upon infection, and it is encountered in
children in the first years of life (between the second
and third years). The pathological process is gener-
ally located in the lumbar enlargement of the spinal
cord—more rarely in the cervical portion. As a re-
sult of the acute inflammatory process the affected
anterior horn undergoes atrophy and shrinking.
Then, among the sequelæ of the disease in the spinal
cord, the following deserve to be mentioned: degen-
erative atrophy of the corresponding motor nerves
and muscles; disturbances in the growth of the cor-
responding extremities; contractures; and loose
joints.

TABES DORSALIS.

The gray degeneration of the posterior columns, which has been found to be the characteristic lesion in tabes, is easily recognized macroscopically through the overlying soft membranes, and may be still more plainly seen upon the surface of a section, which reveals clearly the symmetrical degenerative atrophy and induration of the posterior columns. These columns, which have an abnormal grayish color, are seen, on the section, to have sunken down to a lower level, and to be narrower than they should be. A similar state of things can be shown to exist in the posterior horns and in the posterior nerve roots. The so-called root zones, Goll's columns in the cervical portion of the cord, and Lissauer's fields are almost constantly affected, whereas the anterior portions of the posterior columns in the lumbar portion of the cord, and the so-called posterior external fields in the cervical and thoracic portions of the cord, are almost always spared. The disease of the posterior root fibres begins at the points where they enter the spinal cord.

In tabes the disease does not confine itself to the spinal cord alone: it involves also the entire nervous system. The evidence in favor of this is to be found in the extensive pathological changes which take place in the peripheral nerves as well as in many of the cerebral nerves (the optic nerve, and the nerves supplying the muscles of the eye).

Tabes occurs much oftener in men than in women, and it develops chiefly in individuals between thirty-

five and fifty years of age. In the vast majority of
cases it is a post-syphilitic disease, in which a sort
of late action of certain toxins may, with some degree
of plausibility, be assumed to be the etiological fac-
tor. The point from which the disease starts (blood-
vessels or intermediate substance [?]) remains un-
known.

The Bones and Joints.

DISEASES OF THE BONES.

HYPERTROPHY OF BONE.

Hypertrophy of bone — *hyperostosis* — originates either from the periosteum (as an external or eccentric hyperostosis) or from the medullary spaces in the interior of the bone (as an internal hyperostosis). In the latter case compact bone tissue takes the place of the normal spongy substance. This change is also known by the terms concentric hyperostosis, osteosclerosis, eburnation.

Diffuse hyperostosis and sclerosis are often encountered in the skull cap; the bone in this locality being then twice or even three times as thick as it normally should be, and its spongy interior being converted into compact bone substance. As causes of this change, disturbances in the circulation (stases), rachitis, and syphilis have often been demonstrated.

The external variety of hyperostosis is observed either in a diffuse form, involving the entire bone, or in that of a sharply limited outgrowth—an exostosis.

An exostosis, which is often the product of an ossifying periostitis, is generally composed of compact bone tissue. In a smaller number of cases the interior is found to be spongy. It presents itself under a great variety of forms: serrated, thorn s

often wart shaped or pedunculate, and occasionally ring shaped (a periostosis).

Multiple symmetrical exostoses (developmental exostoses) are uniformly located near the lines of the epiphyses. They grow out from the intermediary cartilage, and they rarely commence their development during youth. When the growth of the normal bone ceases, these exostoses also stop growing. They are apt to be associated with disturbances in the longitudinal growth of the bones. As regards their etiology, heredity undoubtedly plays the leading part. The anomaly is observed more often in men than in women.

Osteophytes are to be considered as constituting special forms of hyperostosis. They are usually distributed over quite large areas. Numerous osteophytes often develop upon the internal surface of the anterior part of the skull cap—particularly during pregnancy. They give to the bony surface a satin-like appearance, and they are usually absorbed during the lying-in period. It is rare that they leave any permanent thickening of the skull. The bodies of the vertebræ are often the seat of osteophytic growths of a more pronounced character—growths which present a serrated, or a wart-like, or a thorn-shaped configuration.

By the term *synostosis* is meant the bony fusion of bones which under normal conditions are separated by sutures.

ATROPHY OF BONE.

Two forms of atrophy of bone are distinguished: the external or concentric, and the internal or eccentric.

The internal atrophy of bone (osteoporosis) is associated with rarefaction of the bone tissue, and is often the result of rarefying osteitis.

Senile or physiological atrophy of bone (osteoporosis) is characterized by rarefaction of the bone tissue, by a partial disappearance of the spongy portion, by a conversion of the compact substance into bone of a spongy character, by dilatation of the medullary spaces and bone canals, and by an increased fragility of the bone-tissue in general. Furthermore, the bone marrow as a rule presents a jelly-like consistence. An analogous condition of osteoporosis often develops as a result of those chronic diseases which induce a general cachexia—such, for example, as cancer and the severe forms of syphilis.

Very similar are the conditions which characterize that form of bone atrophy which results from lack of use (inactivity atrophy), and which is observed in connection with paralyses of various kinds—as, for example, in severe functional disturbances of the joints, and in tabes (in which also it is the paralysis that is the important factor). Such atrophied bones weigh much less than they normally should, and they show diminished powers of resistance and a special tendency to become fractured.

Local atrophy of the bone, due to pressure or wearing away of its substance, occurs, for example, in connection with a neighboring aneurism or new growth.

FRACTURES OF THE BONES.
(Plates 38 and 39.)

The term *traumatic fracture* is employed when the bone breaks in consequence of a force applied from without; while a *spontaneous fracture* is held to be one in which the bone breaks under the influence of a minimal force (entirely inadequate to cause the fracture of a normal bone).

Spontaneous fractures, for example, owe their origin to processes which render the bones brittle. Such are malignant and destructive tumors (carcinoma, sarcoma); inflammatory processes (osteomyelitis, tuberculosis, syphilis, caries, necrosis); and, finally, rachitis and osteomalacia.

Traumatic fractures are either subcutaneous (*i.e.*, simple, or not complicated), without a simultaneous injury of the overlying skin and soft parts; or complicated (*i.e.*, open), as when the point of fracture, owing to the injury of the skin and soft parts, communicates with the outer air.

The fracture is *incomplete*, or constitutes a *fissure*, when the line of the break is a mere crack in the bone, while its outer form remains undisturbed. Such fractures are quite common in the skull cap. In the group of incomplete fractures belongs the condition which is termed *infraction* (green-stick fracture). In this condition the bone is only bent, in very much the same way as an elastic quill becomes partially fractured when too much force is used in bending it.

The *complete fractures of bone* are divided into *transverse, oblique, longitudinal*, and *spiral fractures*, accord-

ing to the direction in which the line of the break runs. When small fragments are separated from the main body of the bone at the point where the break has occurred, it is customary to employ the term *splintered fracture*.

A *direct fracture* is one in which the break occurs at the point to which the force was directly applied. In an *indirect fracture*, on the other hand, the break occurs at a point somewhat removed from that at which the traumatic injury was inflicted.

Bone fractures occur most frequently in men of middle age or of advanced years; such an accident being greatly favored by the condition of senile osteoporosis. In youthful individuals, on the other hand, one often encounters instances of a traumatic separation of the epiphyses.

The alterations which take place at the point of fracture immediately after the escape of blood into the soft parts and the infiltration of these with serum and cellular elements—alterations which constitute the first step in the healing process—consist in the formation of a spindle-shaped tumor, which at first has a soft consistence, but later assumes a certain degree of toughness. This tumor-like mass of tissue is the so-called *callus*. It is customary to distinguish three kinds of callus, in accordance with the differences in the localities from which it develops. The first kind is the *external* or *periosteal callus*, which surrounds the fractured ends of the bone like a ring. It is the product of an ossifying periostitis. The second kind is the *inner* or *myelogenic callus*, which unites the two sections of the medullary canal. And, finally, there is the third kind, the *intermediary callus*,

which is located between the other two, develops from the fractured ends of the bones, and is very scanty in quantity.

The more delicate changes which take place in the development of a callus are the following: In the first place, the cellular elements—both those of the bone marrow and those of the inner layer of the periosteum —commence to proliferate. The cellular and very vascular granulation tissue which is thus produced aids in bringing about a partial absorption of the bone at the fractured ends. In the layer of tissue which lies immediately next to the old bone, quite large epithelioid cells (osteoblasts) are developed, and it is from these that the new bone tissue, fully provided with bone corpuscles, is produced; or else the proliferating periosteum produces either an osteoid tissue in which the bone cells are arranged in groups, or a tissue which consists of hyaline cartilage and which later becomes converted into bone tissue.

The callus which has thus been created is at first rather spongy in character, but later it grows more and more dense; and, besides, it may undergo a still further diminution in size through the absorption of superfluous parts. Indeed, at the last, it is possible for a sort of medullary canal to form.

The formation of a callus often shows—especially in cachectic patients—a tendency to progress slowly. It is considered an unfavorable termination of a bone fracture when the union of the two fragments is effected by masses of fibrous tissue and not by a bony callus. Such a termination is observed in cases of general cachexia and of syphilis. When an imperfect consolidation takes place, there may be formed, at the

point of fracture, what is termed a false joint (pseud-arthrosis). Other local causes may also bring about the same unfavorable termination. Thus, for example, it may be effected by unduly strong pressure upon the point of fracture, especially in cases of complicated fracture; by the interposition of soft parts between the ends of the fragments; by imperfect contact between the opposite surfaces of the fractured ends; and by imperfect immobilization of the parts. As regards the other complications of a bone fracture, the most important is fat embolism, which occurs quite regularly. The semifluid, broken-down bone marrow is taken up by the torn and gaping veins, and through them is conveyed, in the form of drops of fat, to the right side of the heart. From this point it is carried to the lungs and, to a certain extent, to the peripheral portions of the body—to the kidneys, the brain, and the muscular tissue of the heart. When the bone is seriously injured and a good deal of destruction takes place, this fat embolism may favor or be the actual cause of a fatal termination in the course of the first few days after the occurrence of the injury.

RACHITIS.

(Plate 40.)

Rachitis is a disease of growing bone—a disturbance of the metabolic changes which leads to a diminished deposit of calcareous material. Fundamentally the disease consists in an excessive formation of osteoid tissue—i.e., of the material which constitutes the groundwork of bone—without an adequate deposition of chalky material. In the long tubular bones the

both the bones and the soft tissues, in these cases, showing a diminution in the amount which they contain not only of lime but also of those elements which remain as ashes after everything else has been destroyed by fire.   In this form of rachitis all the pathological alterations disappear just as soon as sufficient (and suitable) nutriment is supplied to the tissues. A somewhat similar form of the disease—that is, one that is dependent upon the lack of lime—may occur in children when, notwithstanding the fact that their nutriment contains the requisite amount of lime, its absorption into the system is materially diminished by the long persistence of digestive disorders—more especially diarrhœal affections.   A tendency to certain diseases—*e.g.*, bronchitis, catarrhal pneumonia, catarrhal affections of the stomach and intestines, and spasm of the glottis—is often a characteristic of rachitis.   The ultimate cause of rachitis is not clearly understood.   The artificial nourishment of infants with unsuitable articles of food, and disturbances of the digestive organs, as well as unfavorable hygienic surroundings, undoubtedly favor the development of this disease.   On the other hand, as a rule, it only rarely attacks children who derive their nourishment from the breast.

That rachitis is favored by living in confined quarters, by breathing bad air, and by lack of physical

Fig. 7.—Curvature and Deformity of the Lower Extremities, Due to Rachitis. In this patient—a girl, eight years old—who had suffered severely at some previous time from rachitis, but in whom the disease might at this time be considered as cured, the tubular bones, and especially those of the left side, are curved in a marked degree and in various directions, and at more than one point they are sharply bent (partial fractures). The upper extremities are only very slightly altered.

FIG. 7.

exercise, is most clearly shown by the fact that during the winter months the disease progressively increases in frequency, reaching its maximum in the spring and early summer, and its minimum in November and December.

The so-called fœtal rachitis (chondrodystrophia fœtalis) has, in the majority of cases, no relationship whatever to true rachitis; it is, in fact, a peculiar and variable anomaly in the development of the cartilaginous structures.

OSTEOMALACIA.

(Plate 41.)

Osteomalacia occurs only in adults; it is a disease of fully developed bone. The Haversian canals and the medullary spaces are surrounded by a broad ring of tissue containing no lime. Through the melting down of tissue adjoining lacunæ coalesce and fissures form between the fibrillæ. Atrophy of the bone tissue goes hand-in-hand with the gradual disappearance of its calcareous elements, and with this loss absorption of the bone cartilage is also associated. The newly formed fissures and canals become filled with lymph or marrow fat; they become medullary spaces, and it is to their presence that bone affected with osteomalacia owes its porosity. This process is confined largely to individuals of the female sex. It often begins during one period of pregnancy, and then becomes aggravated during each succeeding period. The entire skeleton grows smaller. By reason of their rubber-like softness the individual bones become curved and bent in a variety of ways. The order in

which they are attacked is the following: first, the
pelvis; then the spinal column and perhaps one
shoulder blade; and, finally, the extremities and the
other shoulder blade.

From a chemical analysis it is learned that either
no increase whatever, or only a moderate increase,
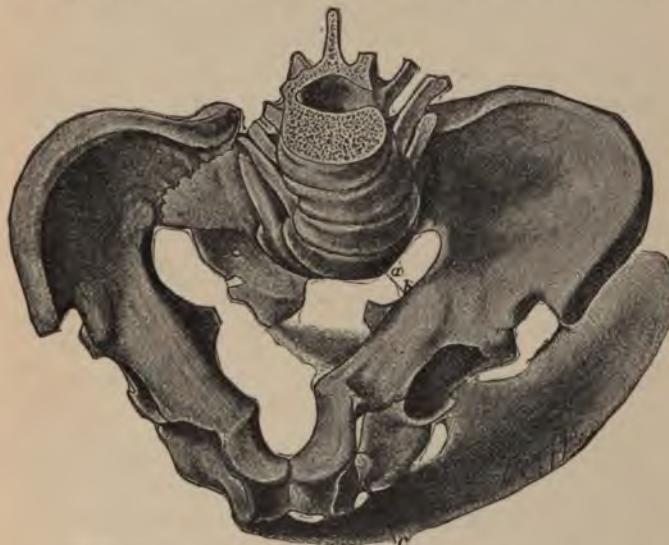
Fig. 8.—Osteomalacia of the Pelvis (one third natural size). A pelvis af-
fected with osteomalacia of non-puerperal origin. While at some earlier
date it had been in a softened condition, at the present time it has once
again become entirely ossified; *a*, *b*, connecting line between the middle
of the fourth lumbar vertebra and a point in the linea innominata just
above the acetabulum; *c*, an exostosis, about the size of a cherry, on the left
ramus ascendens ossis ischii. (Preparation belonging to the Munich
Gynæcological Clinic.)

takes place in the gelatin-yielding substance of the
bones; but that, on the other hand, the total salts
contained in them are markedly diminished—to the

extent of about eighteen per cent. This diminution appears to be greater in the spongy than in the compact substance of the bone—a difference which favors the idea that the pathological process advances from the medulla toward the periphery and in the direction of the epiphyses.

In quite recent times the belief has found favor that osteomalacia is a trophoneurosis of the bones, which has for its starting-point the ovaries. Such a belief makes it easier for us to understand the connection between the process in question and ovulation, pregnancy, and the lying-in period. Of special interest, therefore, is the fact that the operative removal of the ovaries has effected a large number of cures (eighty per cent) of osteomalacia, whereas formerly the mortality of the disease amounted to precisely this figure, viz., eighty per cent.

ACUTE OSTEOMYELITIS.

This disease begins, as a rule, in the neighborhood of the epiphyses of the long bones, but it does also develop, in a few cases, near the spot at which an injury, a fracture or a shot wound, has occurred at some earlier date. The disease is confined almost entirely to youthful individuals who have not yet ceased to grow. So far as its essential nature is concerned, it is a disease due to infection—a hæmatogenous and cryptogenous infection by staphylococci. In rare cases it may be produced by other micro-parasites (streptococci, typhoid bacilli, pneumococci). It is not known exactly how these carriers of infection find their way into the body. Now and then the osteo-

myelitis seems to develop as a sequence to some trivial suppurative process. The bones most often affected are the tibia, the femur, and the humerus, while the other tubular bones of the extremities are comparatively seldom involved.

At first there is a stage of hyperæmia, accompanied or not, as the case may be, by small hemorrhages; then follows the transformation of the fatty marrow into lymphoid tissue; and this, in turn, is soon followed by a constantly increasing infiltration of the latter with pus corpuscles, until it assumes a grayish-white or yellowish color. Ultimately purulent foci develop in this transformed marrow. While in this stage the disease often ends in death, with the symptoms of an acute septicæmia or septico-pyæmia; or it may involve the surrounding bone substance, and lead to its disintegration—i.e., to caries and necrosis, with the formation of sequestra and the establishment of a fistulous channel through which a small sequestrum may be expelled. Very often subperiosteal abscesses develop in connection with the disease; and still another accompanying phenomenon may be the production—proceeding from the periosteum—of a number of osteophytes or of a mass of bone substance, which surrounds the sequestrum like a box splint. Toward the healthy end of the bone an ossifying osteomyelitis takes place, and this produces a condition of sclerosis of the medullary cavity; the destructive disease being in this manner shut off, as it were, from the rest of the body.

In acute osteomyelitis the rarefying osteitis may sometimes lead to the production of deformities, but it rarely leads to softening and curvature of the bone.

If the disease is located near the epiphyseal line, the epiphysis may become loosened from its attachment to the underlying bone, and the intermediate cartilage may either undergo simple displacement or else be destroyed by suppurative inflammation.

TUBERCULOSIS OF BONE STRUCTURES.
(Plates 46 and 47.)

Tuberculosis of bone occurs rarely as a primary disease; it is almost always secondary to tuberculous disease in some other part of the body. The infection is uniformly hæmatogenous in character; that is, the tuberculous poison is transferred from one spot to another along the route of the blood current, in much the same way as an embolus is transported. Youthful individuals are specially predisposed to the disease. The soft parts of a bone—particularly the bone marrow—undoubtedly afford, for the lodgement and multiplication of the poison (the tubercle bacilli), conditions as favorable as those furnished by the lymph glands. Tuberculous disease of bone or of a joint develops very often as a result of some mechanical injury; and this circumstance explains how it happens—which is a fact—that the male sex is more afflicted with this form of tuberculous disease than is the female. (Differences in the injuriousness of their respective occupations.) So far as the question of localization is concerned, it is sufficient to state that the bones of the trunk of the body (more particularly those of the spinal column) are affected in about one-third of all the cases, and those of the lower extremities are affected in about the same proportion; while

the bones of the upper extremities are involved in a little more than one-fifth (twenty-two per cent) of the cases.

Tuberculous osteitis and osteomyelitis begin most often in the epiphysis of a tubular bone. As a result of the tuberculous infection there is developed a granulating and rarefying inflammation, which causes the bone substance to break down and melt away. The granulation tissue, which contains miliary tubercles, giant cells, and tubercle bacilli, destroys by erosion the bone trabeculæ, and at the same time it manifests a tendency to undergo itself cheesy necrosis. This granulating and cheesy inflammation quickly leads to extensive destructions of bone tissue, and these larger and smaller foci in which the bone has been destroyed show a tendency to break through to the surface (as into the cavity of a joint or out toward the periosteal covering of the bone). In the interior of these cavity-like defects in the bone, pus and cheesy material, more or less intermingled with spongy sequestra, are found; and the walls of these excavations are lined in many places with granulations mingled with pus and cheesy material.

The periostitis which develops secondarily, at a later stage of the disease, leads to the accumulation of cheesy and purulent matter, and often to the formation of an abscess (the so-called burrowing abscess), particularly on the anterior surface of the body of a vertebra. Necrosis usually follows after the bone is deprived of its periosteum, or else fistulous channels form in the soft parts and carry pus from the surface of the bone out upon the surface of the skin.

In many cases there will develop, *e.g.*, on the sternum or on the ribs, a tuberculous periostitis and osteitis; and in such cases the disease can be traced to a tuberculosis of some neighboring organ (pleura or lymph glands), from which it has spread to the new locality by direct continuity. In a similar manner a primary tuberculosis of a joint may spread by direct continuity to the neighboring bone structures.

An ossifying periostitis is often associated with tuberculous disease of bone, and in that event—as happens in purulent osteomyelitis and osteitis—it is very likely to produce an outside casing of bone, in which there are one or more fistulous openings that afford an outlet to the more deeply situated pus.

A spontaneous cure of a tuberculous bone caries takes place rarely. It may be greatly promoted, however, by surgical interference—by the removal of sequestra and diseased tissues.

The term *spina ventosa* is employed to designate a form of fungous osteomyelitis (also of a tuberculous nature) which, as a local process, often primarily attacks the short tubular bones of the extremities (the phalanges). In these cases the entire shaft of the bone is involved. The production of granulation tissue in the marrow is associated with a breaking down of the central bone substance and with a certain degree of expansion of the bone, which often becomes as thin as paper and eventually disappears altogether. Thus an escape is provided for the accumulated cheesy and purulent masses.

SYPHILIS OF BONE.

Bone syphilis is characterized by the chronic course which it pursues. The periosteum is the part most often affected. In simple syphilitic periostitis— which localizes itself principally in the deepest layers of the periosteum, and is encountered especially on the skull and on the tibia—the periosteum first increases in thickness and then produces osteophytes or irregularly shaped nodular growths of bone tissue. In the gummous form of periostitis semisolid, almost fluctuating nodules are produced. Periosteal gummata like these may be entirely absorbed. In quite a number of cases the underlying bone may be worn away to a greater or less extent, or it may even become perforated (the so-called caries sicca). When the periostitis results in a breaking down of the tissues (suppurative periostitis), the inflammation may extend to the adjacent and superjacent soft parts. The discharge from such a focus of disease consists of a slimy, ropy mucus, mixed with blood and pus. The so-called cold abscess (with a tendency to burrow) may also form under these circumstances. Finally, the disease may end in caries and necrosis of the bone, and cicatricial tissue may form at the spot. The cicatrix of the overlying skin is generally drawn inward, by reason of its close connection with the bone cicatrix.

The simple and gummous forms of periostitis find their counterparts in the simple and gummous forms of osteitis and osteomyelitis. The former of these displays a proliferative tendency, which leads to the

development of hyperostosis in the affected bone, whereas the latter shows a tendency to develop deposits in separate foci or centres. The disease is considered to be pursuing an unfavorable course when separate centres of necrosis develop and sequestra are formed. The bone undergoes rarefaction, and shows (especially in the skull) not only quite large defects, but also a worm-eaten appearance. In the case of the tubular bones a rather thick shell of bone sometimes forms around them. The bony walls surrounding the sequestrum appear to be thickened and very uneven on their external surface.

The bones which are located near the surface of the body seem to be specially liable to be affected with syphilitic disease. The following are favorite seats of the disease: the bones of the skull cap (particularly the tabular part of the frontal bone and the parietal bones); the bony framework of the nose, and the hard palate; the diaphyses of the tubular bones —especially of the leg and of the forearm; and, finally, the clavicle and the bodies of the vertebræ.

The bones of the framework of the nose are often secondarily involved in cases of syphilitic ulceration of the mucous membrane. When in this way the bone is left bare, it is likely to undergo necrosis. Then, again, a primary gummous periostitis and osteitis may attack the bones of the nose, in which case the necrotic bone fragments become loosened and are eventually cast out. When the necrotic process covers a wide extent, the nose may be deprived of its bony support; and when the cribriform plate of the ethmoid is destroyed, the bridge of the nose sinks in, like the seat of a saddle. Oftentimes the necrosis at-

tacks also the nasal bones proper, and then it may
extend until the nasal process of the superior maxilla
and the horizontal plate of the palate bone, or the
palatine process of the superior maxilla, are involved
in the destructive disease. It is in this way that de-
fects in the hard palate are produced. A necrosis of
the lamina cribrosa of the ethmoid bone has some-
times given rise to a secondary purulent meningitis.

ACTINOMYCOSIS OF BONE.

(Figs. 9 and 10.)

The alterations which characterize this disease are
similar, in their general character, to those which
occur in tuberculosis. Usually there is a combina-
tion of fungous osteitis with suppuration, the results
of which are extensive melting down and necrosis
of bone tissue, with enlargement of the bone itself
(rarefying osteitis); the production of osteophytes;
and the diffuse proliferation of those layers of bone
which are in close proximity to the disease (ossifying
osteitis and periostitis).

PHOSPHORUS NECROSIS.

This disease attacks, with special frequency, the
jaw bones. At first, a small abscess (a parulis or
gumboil) develops in the neighborhood of a carious
tooth, or it may also develop in a jaw the teeth of
which are perfect. Gradually the teeth near this ab-
scess become affected; they appear to be loose in
their sockets. As a result of the persistent tendency
to suppuration, new abscesses are constantly being

The [illegible] of the jaw-like. (After Illisk.) In the region [illegible] nodular prominences, most of [illegible] reddened skin, which is abnormally [illegible]. It a few places the skin has be[illegible] to [illegible] figure that it permits the underlying yellowish [illegible] through it, at spots which are about as large as a millet [illegible]. In the neighborhood of these nodules the soft parts are the seat of [illegible] infiltration. Palpation, by means of the [illegible] mouth, reveals the fact that the cheek [illegible]. Some months before the disease first showed [illegible] had a carious molar tooth extracted from the upper [illegible] toward the front. Two months later there ap[illegible] of the cheek, at a point corresponding to that

where the tooth had been extracted, a tumor about the size of an English walnut. It felt hard, and was at first painless in character, but later it became somewhat sensitive. Then, soon afterward, the whole cheek became swollen; and in the midst of this hardened area of the cheek there developed, at a later period, several tough and prominent tumors, which gradually assumed a somewhat softer consistence; no thickening of the jaw-bone itself. Under suitable surgical treatment (incision; thorough scraping of the wound; opening up and scraping of the fistulous tracks) a cure was effected. The patient was a stable boy, eighteen years old. (Compare A. Illich: "Beitrag zur Klinik der Aktinomykose." Wien, 1892. Case V., on page 7.)

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formed, and these in time break, either into the cavity of the mouth or outwardly through the swollen and hard tissues of the cheek. In the latter case, fistulæ are formed, which open on the face or on the neck, or—where the upper jaw is diseased—beneath the margin of the orbital cavity. In addition to the inflammatory swelling of the soft parts there may be, as a result of proliferative processes in the periosteum (toxic ossifying periostitis), a certain amount of thickening and expansion of the bone. At first, small sequestra may be cast off, and the discolored necrotic bone may be exposed to view; then the alveolar process, having completely died, projects into the cavity of the mouth; and so, in the course of a few months, the entire lower jaw may be completely destroyed by necrosis. The new tissues thrown out by the periosteum lie like pumice stone upon the old bone, and in course of time they also, in their turn, may be destroyed by necrosis. After the necrotic portions of bone have been cast out, or have been removed by surgical interference, the defect may in great measure be filled in by a new formation of bone on the part of the periosteum, or by the growth of granulation tissue. When the suppuration ceases, the fistulous channels heal and become closed. Markedly re-

Fig. 10.

tracted cicatrices and deformities of different kinds remain as permanent results. Phosphorus necrosis is not a local affection, but rather a general disease of the osseous system. It develops, as a secondary process, in bones which have already previously been somewhat altered by the toxic influence of the phosphorus. The disease attacks not only the jaws, but also the bones of the extremities, which then become thickened (hyperostosis) and abnormally brittle. (It

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FIG. 10.—Multiple Caries and the Development of Osteophytes in the Thoracic Portion of the Spinal Column and in the Ribs, in a Case of Actinomycosis. (After Ponfick.) At the post-mortem examination there was found a retromediastinal abscess cavity, which extended from the second to the eleventh thoracic vertebra. The anterior surface of the bodies of the vertebræ was bare at many spots, and revealed to view a great variety of granular and dentated elevations and outgrowths. Between these there were areas whose uneven surface was covered with a thin stratum of brownish granulations. The intervertebral discs were found to be superficially eroded, and already in a state of beginning disintegration.

In the intercostal spaces it was found that the pus had penetrated far in among the bands of muscular tissue.

In this patient—a man, forty-five years of age—the disease had begun as an inflammation of the costal pleura, on the left side; but, as time went on, he did not seem to recover from the effects of this inflammation. Six months before his death he complained of quite sharp pains and other uncomfortable sensations in the region of his back and loins; and soon afterward abscesses formed at different points. Some of these broke directly through the skin of the back, while others burrowed extensively underneath it. There were at this time marked symptoms of a slowly increasing thickening of the pleura, low down on the left side. Terminal exudation into the pleural cavity of the right side, with beginning pericarditis. As stated above, a prevertebral phlegmon was found, at the autopsy, in the posterior portion of the mediastinum. There was also a parapleuritic cavity which extended, on both the right and the left sides, from the seventh to the ninth intercostal space, and which communicated with the outside world through a large number of old fistulous channels in the substance of the longissimus dorsi muscle and also in that of the muscles belonging to the shoulder blades. These fistulous channels also ran in various directions through the subcutaneous connective tissue of the entire back. Evidences of a recent fibrino-purulent pericarditis were also found. (Compare E. Ponfick: "Die Aktinomykose des Menschen." Berlin, 1882. Case I., on page 7.)

is not an uncommon thing to observe numerous frac-
tures before the necrosis sets in.)   After the bone has
been subjected to the toxic influence of the phos-
phorus, a septic infection must then first take place
before it can become necrosed.   Whereas formerly
the mortality from phosphorus necrosis was very large
(from twenty-five to fifty per cent), it is now possible
in almost every case to effect a cure provided early
operative measures be resorted to and the necrosed
bone be all removed.

NEW GROWTHS OF THE BONY STRUCTURES.

(Plates 48 and 49 and Figs. 11–15).

Malignant growths of all kinds—particularly can-
cer and sarcoma—occur in bone, either through me-
tastasis or as a secondary manifestation.   Now and
then a case is seen in which the bone marrow and the
bone tissue itself are infiltrated with genuine cancer
elements; and when this happens the bone becomes
bent and deformed in very much the same way as it
does in osteomalacia.   This condition has been desig-
nated as an osteomalacia carcinomatosa.   Now and
then the bone becomes involved secondarily by the
advance of the destructive new growth, from some
point near by, into the bone structure.   Cancerous
disintegration of the bone soon follows.   This sort of
invasion is apt to occur in cases of cancer of the skin
(on the face and on the extremities; see Plate 49), or
of sarcoma involving some portion of the cavity of the
mouth or the gums.   In such cases the bones of the
jaw often undergo partial destruction.

Among the *primary new growths* which develop in

FIG. 11.—Fibroma of the Lower Jaw. On the left half of the face there is a tumor as large as a child's head, which is of a very tough consistence, and stands out about ten centimetres above the normal level of the cheek. It extends from the lower margin of the orbital cavity and the zygoma downward as far as to the middle of the neck. The skin, which is tensely stretched over the almost immovable tumor, can still be moved a little over the surface of the growth. The mouth is displaced toward the right side. The lower jaw can be separated to only a slight extent from the upper jaw. The posterior third of the horizontal ramus and the entire ascending ramus of the lower jaw have disappeared completely in the mass of the tumor. When pressure is made upon the growth a semifluid, bad-smelling, abnormally colored material escapes into the mouth from a point situated between the second and third molar teeth. Resection and exarticulation of the lower jaw were performed, and a complete cure followed. An examination made after the removal of the diseased jaw revealed the following conditions: In the interior of the tumor, which consisted of fibrillated connective tissue, there was a cavity, about the size of a goose's egg, which was filled with pus and detritus; and from this

cavity a fistulous channel led to the margin of the gum, where it opened behind the second molar tooth. The larger part of the bony structures of the jaw had been supplanted by tumor tissues. The patient, a man about thirty-three years of age, stated that the tumor had been growing during a period of four years, and that the first evidences of its existence were discovered on the gum surrounding the left lower wisdom tooth. (For a more detailed description of this case consult the dissertation of Alfred Sternfeld: "Ueber Exarticulation des Unterkiefers zur Entfernung von Tumoren." Munich, 1880.
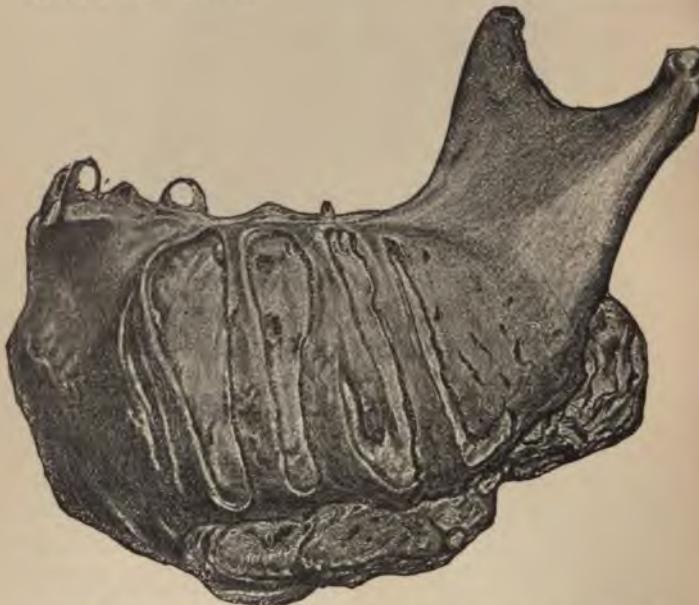
Fig. 12.—Central (enosteal) Fibroma of the Left Lower Jaw. (From a boy, ten years old.) Upon the outer surface of the growth there are several parallel ridges of bone which run in an up-and-down direction, and between them the grayish-white tissue of the growth protrudes. The spicules of bone appear to grow out from the jaw. The inner medial surface of the tumor is quite flat and shows no traces whatever of bone formation.

The tumor grew from the connective tissue of the bone, and not from the periosteum. Its advancing growth not only caused the bone of the jaw to be absorbed but it also excited inflammatory action on the part of the periosteum. It was through the latter inflammation that the shell-

like envelopes of bone came to be formed; and these, in their turn, as the growth of the tumor advanced, were to a certain extent absorbed. The spaces separating the parallel ridges of bone on the outer side of the tumor indicate the spots where this shell of bone was completely absorbed. The tumor has a very tough consistence, and creaks when cut with the knife. It is knobbed and lobulated in its structure. Under the microscope it shows a fibrillated tissue, containing very few cells and blood-vessels.

At first, the fact that this was a tumor, or new growth, was not appreciated; it was supposed to be a simple periostitis. Two years ago the patient had an attack of mumps, and from that time onward the growth increased slowly in size. Seven months before the operation was performed a tooth was extracted, with the expectation that this would facilitate the discharge of the pus which was supposed to be confined within the swollen tissues. Finally, resection and exarticulation of the diseased jaw were performed (by Privatdocent Dr. von Stubenrauch), and the wound healed in the course of three weeks.*

Fig. 13.—Multiple Chondromata of the Fingers. (From a child.) On the fingers of the left hand—with the exception of the thumb and little finger—there is quite a number of roundish, hard nodules, which in some places are more or less fused together, and which consist of cartilage tissue.

* A more detailed description of this case will be found in the dissertation of Gottfried Kentenich, entitled: "Ueber Kieferfibrome." Munich, 1896. (This dissertation contains the complete statistics of all the cases of fibroma of the jaw which have thus far been observed.)

Fig. 14.

bone structures, *sarcoma* is the commonest. *Fibro-mata, osteomata*, and *chondromata* occur more rarely. The primary bone sarcomata develop either from the medullary substance or from the periosteum. 1. *Central* or *myelogenous sarcomata* (Plate 48). In this category belong the *giant-cell sarcomata*—tumors which are fairly benign, light brown in color, and generally surrounded by a shell of bone; and also the soft and marrow-like *round-cell* and *spindle-cell sarcomata*, which

FIG. 14.—Multiple Enchondromata of the Shoulder Blade and Humerus. (Two-fifths natural size.) (Case of Steudel.*) In this case (a man, forty-five years of age) a number of enchondromata developed in the shoulder blade and in the epiphyses of the humerus. As a result of the presence of so many outgrowths of different sizes, the scapula was a good deal deformed. The humerus was considerably shortened, and both epiphyses were thickened and knobbed, whereas the diaphysis, throughout a length of eight centimetres, was of normal thickness and presented a smooth surface. The individual tumors were of a soft and elastic consistence and presented a knobbed and uneven surface.

Cartilaginous tumors of a similar character were found, in another case (a man, twenty years of age), upon both thighs and legs; and upon the fingers and toes, particularly of the right side, these growths were very numerous—so much so, in fact, that it was found necessary to amputate the right forearm. In addition to those already enumerated there were many tumors on the skull, on several of the ribs, on the spinal column, and on the pelvis. The only parts of the body which were entirely free from these growths were the upper arm and forearm of the left side. A microscopic examination showed that these tumors were composed of cartilage of a purely hyaline character. The first evidences of these tumors were discovered when the boy was between six and seven years of age. By the time he had reached his twentieth year their development upon the extremities had ceased. Numerous other anomalies were observed on the bony structures of the body, some of which had increased very greatly in size. Thus, for example, there were curvatures of the extremities and of the thorax; shortening of the affected bones; and numerous fractures, which had resulted from trivial mechanical injuries, and which were due to the abnormal softness of the entire skeleton. Finally, in addition to these pathological alterations, multiple cavernous angiomata, growing out from the walls of the veins, were found in various parts of the body.

* E. Steudel: "Multiple Enchondrome der Knochen"; in Bruns' "Beiträge zur klinischen Chirurgie," Bd. viii., 1871.

FIG. 15.—Sarcoma (fibro-myxo-sarcoma) of the Tarsal Bones of the Foot. The larger part of the foot and the lower portions of the leg are occupied by an irregularly shaped, nodulated tumor, the surface of which has become ulcerated at different points. The growth takes its origin from the tarsal bones. The patient, a man, forty years old, states that the tumor began to show itself two and a half years previously, after his foot had received some kind of an injury. Amputation of the leg. (No. 255 in the *Einlauf-Journal*, 1882.)

grow in an irregular fashion and at an early stage eat
their way through the bone. 2. *Periosteal sarcomata.*
These grow from the inner layers of the periosteum,
which then surrounds the tumor like a fibrous cap-
sule. The destruction of the bone, in these cases,
takes place from without inward. These periosteal
sarcomata, which are usually of the spindle-cell vari-
ety, are of a more malignant character; they are often
so soft as to fluctuate, and they show a greater ten-
dency to produce metastases. A bony framework is
often developed in these tumors through the conver-
sion of the newly formed connective tissue or cartilage
tissue into osseous tissue. Such growths are desig-
nated as *osteo-sarcomata.*

DISEASES OF THE JOINTS.

LUXATION.

(Plate 50.)

The terms luxation and dislocation are commonly
understood to mean an anomalous condition of a joint,
of such a character that the articular end of one bone
fails to come in contact with the articular end of an-
other (the associated) bone, and protrudes through a
rupture in the articular capsule. When only certain
parts of the capsule and ligaments are torn, while at
the same time the articular ends of the bones remain
in contact, it is customary to designate the lesion as
a distortion or a sprain.

Of the two parts involved in a luxation or disloca-
tion that which lies nearest to the periphery is spoken
of as the dislocated part. Thus, for example, in a

dislocation involving the shoulder-joint, it is the humerus which is the part dislocated; that is, there is a dislocation of the humerus.

Dislocations are of three kinds: Congenital, spontaneous, and traumatic.

A *congenital dislocation* (Fig. 16) which occurs, for example, in the hip-joint depends upon defects in development which have originated during intra-uterine life (*e.g.*, hypoplasia of the acetabulum), or upon displacements that have taken place during the same period (fœtal dislocations of a spontaneous nature). Dislocations of this kind occur most frequently in the female sex (eighty-seven per cent), and in two-fifths of all cases both sides of the body are affected.

Spontaneous dislocations occur as a result of articular inflammations. They may be due to relaxation and softening of the ligaments and surrounding capsule—alterations which may occur in the exudative forms of inflammation. (These are termed dislocations due to distention.) Or the luxation may occur in consequence of a destructive caries and necrosis of the articular ends of the bones, this being especially apt to happen at the hip and knee joints. (These are termed dislocations due to the destructive action of disease.)

Traumatic dislocations are rarely the result of violence inflicted directly upon the joint; they are produced, in the great majority of instances, by a force which acts upon the latter indirectly. In the indirect form of luxation the dislocated end of the bone is pushed with such force against the articular capsule as to cause it to tear and so permit a passage for the bone through the torn opening. The articular end of

this bone fails, then, as a matter of course, to come in contact with the corresponding end of its opposite neighbor. In such a dislocation the outlines of the joint are altered—that is, the latter is deformed—and often the nerves and blood-vessels, as well as the soft parts in the neighborhood, are more or less injured. If, at the same time, there should be a fracture of the bone with a wound of the skin extending down to the seat of the fracture, it is customary to speak of the condition as a *complicated fracture.*

In a favorable case, after the parts have been restored to their natural positions, a process of regeneration sets in; the torn capsule heals, the effused blood is absorbed, and, at the end of from one to two weeks, the joint is once more able to perform its functions. In an unfavorable case, if the parts cannot be restored to their natural positions, a new joint forms on the head of the permanently dislocated bone; and, from the periosteum of the bone against which the latter rests, there is often developed a wall-like mass of new bone, which, in a somewhat imperfect manner, supplies the requirements of a new joint socket (Plate 50). In rare cases a *fibrous or bony ankylosis* may take place; and when this happens the dislocated bone becomes firmly anchored to its new bed and to the surrounding parts by newly formed bone tissue or connective tissue.

In what is known as *habitual dislocation* there seems to be a predisposition to frequent repetitions of this accident—oftentimes under very slight provocation. The reduction of the dislocation, in these cases, is generally an easy matter to accomplish. Among the causes of this habitual luxation may be mentioned

FIG. 16.—Congenital Dislocation of Both Hip-Joints. The patient is a
girl, four and one-third years of age. The lower extremities appear to be
shorter, and the region of the hips broader, than normal. There is
well-marked lordosis, which disappears entirely when the child assumes

the horizontal position. If traction is exerted upon both legs, while the
pelvis is held in a fixed position, it will be seen that these increase in
length. Both active and passive movements are easily executed; only
those of abduction and rotation being somewhat restricted. (A more ac-
curate description of this case will be found in the dissertation of Niko-
laus Cicei, entitled: "Zur Kenntniss der Congenitalen Hüftgelenkluxa-
tionen." Munich, 1884.)

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injuries which involve extensive portions of the joint,
and abnormal dilatation (of a permanent character) of
the capsular ligament of the joint.

As regards the localization of traumatic dislocations
it is worthy of remark that ninety-two per cent of them
involve the upper extremity, only five per cent the
lower, and nearly three per cent the trunk.

ACUTE INFLAMMATION OF JOINTS.

This disease occurs in various degrees of severity,
which are characterized by corresponding changes in
the quality of the exudation. The latter is either
serous in character (as in inflammatory dropsy of a
joint), or sero-fibrinous, or purulent.

In *acute articular rheumatism* (polyarthritis rheu-
matica acuta) several joints are almost always affected.
In addition to the presence of swelling and moderate
redness of the lining synovial membrane, the articu-
lar cavity is generally found to contain a sero-mucoid
or a sero-fibrinous exudation in which there are no
bacteria. There is often associated with the disease,
at the same time, an acute endocarditis—evidently of
infectious origin. Those cases which are believed to
be instances of acute febrile articular rheumatism
which end fatally, and in which a purulent exudation
containing pyogenic micro-organisms is found in the

joint cavities of the affected joints, should be classed rather as cases of septic polyarthritis.

SUPPURATIVE ARTHRITIS.

This disease occurs very rarely as a primary affection, and when it does it is as a part of a septico-pyæmia of cryptogenous origin. It is more often a secondary disease, of metastatic and hæmatogenous origin. It occurs, for example, in the course of a puerperal septicæmia, and in ulcerating septic endocarditis; or it may follow an attack of scarlet fever or gonorrhœa. The villi of the synovial membrane appear, in this disease, to be infiltrated with cells; and, when the inflammation is intense, both the endothelium and the cartilage perish through processes of necrosis. In the cavity of the joint a varying quantity of whitish, creamy pus will be found. In many cases the inflammation spreads to the neighboring tissues, that is, to the peri-articular connective tissue, and there sets up a suppurative peri-arthritis.

This suppurative form of arthritis may also develop from an injury to the joint of such a character as to lay it open to the air; and finally it may originate from neighboring bone disease, as is likely to happen when a purulent osteitis or osteomyelitis is going on in the vicinity of the joint. It is then termed an osteo-genous arthritis.

The disease rarely terminates in a cure. The articular ends of the bones, being robbed of their cartilage, begin to develop granulation tissue. It grows out from the synovial membrane and from the bone marrow, and unites with the granulation tissue which

develops from the opposite articular surface. A fibrous metamorphosis then takes place in these inflammatory products and leads to the union of the joint surfaces—*i.e.*, to a fibrous ankylosis, or, if ossification takes place in the newly formed masses of tissue, to a bony ankylosis. The process which takes place in a case of this kind is similar to that which is observed in adhesive inflammations of serous membranes like the pleura and the pericardium.

GOUTY ARTHRITIS; ARTHRITIS URICA.

(Plate 51.)

Under the influence of some disturbance of the general metabolic processes (such as the defective conversion of the uric-acid which is formed in the spleen and in the lymphatic organs of the digestive tract into urea, and the retention of uric acid in the blood and in the liver) uric-acid salts (sodium urate or calcium urate) are deposited in the articular cartilages. This does not take place all at once, but little by little, on different occasions, and while the synovial membrane is in a condition of inflammatory redness; and, furthermore, the deposit of uric acid is preceded by inflammatory and necrotic changes. The products which give rise to these predisposing necrotic changes in the tissues are still, for the present, unknown. The urates are deposited in the cartilage cells and in their capsules in the form of star-shaped needles and bundles of crystals. The cartilaginous covering of the affected joints (particularly the metatarso-phalangeal joint of the big toe and the finger-joints) presents the appearance, in the early part of the disease, as if

Fig. 17.—Gouty Fingers; Polyarthritis Urica of the Finger-Joints. In this case the finger-joints—with the exception of those of the two thumbs—are very much enlarged, and this is especially true of the middle joints. The skin covering these enlarged joints is white and appreciably thinned, so that it is possible to distinguish through it the yellowish-white masses of uric acid. There are several old cicatrices, representing the remains of former openings through which a portion of the accumulated uric acid had escaped. Deposits are also present in the sheaths of the tendons in the metacarpal part of the hand.

Still larger swellings of the joints, of the same character as those observed in the fingers, are present upon the lower extremities, and particularly at the ball of the right foot. Here there is a very large swelling, with several openings, through which, during the past nine months, whitish-colored masses of varying consistence have been discharged. At first, solid concretions, varying in size from that of a bean to that of a hazelnut, and weighing in the aggregate about sixty grams, were cast out; but afterward the discharge which escaped from these fistulous openings was of a pap-like consistence and greasy in character. When allowed to dry it became converted into a mass of substance like mortar. The skin of the entire foot is swollen, of a livid color, and traversed by cord-like veins. Alterations of a similar character (without any fistulous openings) are visible on both knees and at the shoulder-joints; and, besides, there are gouty deposits in both auricles. The patient, a man forty-nine years old, says that he had had the disease for a period of thirteen years; and that, in his capacity of steward of a ship, he had been for many years exposed to all sorts of weather and to hardships of different kinds. Two of the patient's brothers, it appears, are also sufferers from gout (collateral hereditary taint). The patient admits that he is a moderate user of the stronger alcoholic drinks (rum, cognac, punch, and the different liqueurs). (Further details in regard to this case will be found in the dissertation of H. Keiper, entitled: "Zur Kenntniss der Gicht," Munich, 1889.)

a delicate sediment, of a white or milky-white color, had been disseminated throughout its substance. In the severe cases, and in those in which the process has been increasing in severity through a long series of years, this deposit may so increase in quantity as to form knobbed masses (tophi) of a mortar-like or chalk-like material. In the latter event the soft parts covering these masses grow thinner and thinner, until finally the skin gives way and permits at least a part of the pathological concretions to escape spontaneously. The articular surfaces of the bones which compose a joint are not the only parts in which such deposits take place; the enveloping capsule and the adjacent tissues may also become similarly affected. Now and then, in these gouty patients, one finds at the same time evidences of a chronic atrophic nephritis urica ("gouty kidney"). Among the causes which favor the development of the disease should be mentioned an inherited predisposition, chilling of the body, and high living.

TUBERCULOUS ARTHRITIS.

(Plate 52.)

Whereas miliary tubercles, unaccompanied by inflammatory alterations, rarely develop in the synovial membrane, tuberculous disease of the other joint structures—which is characterized, in the great majority of cases, by a tendency on the one hand to produce an exudation (partly free, and partly organized), and on the other to cause destruction of tissue—is by no means a rare occurrence.

Tuberculous disease of a joint usually begins—as

do many cases of tuberculous pleuritis—with the pour-
ing out of a sero-fibrinous exudation. In rare in-
stances this first stage is immediately followed by a
retrogression of all the symptoms and a cure. In the
great majority of cases, however, organization and
vascularization of the deeper layers of the fibrinous
pseudo-membrane soon take place. And while this
is going on, new layers of fibrin are constantly being
deposited on the surface which is turned toward the
cavity of the joint—a process which is accompanied
by the exudation, into the latter, of either a clear
fluid or one that is filled with flocculent material. At
the same time numerous tuberculous nodules develop
in the organized pseudo-membrane, and these nodules
show a decided tendency to fuse together and to break
down into cheesy material. Spongy granulations
clothe more or less completely the internal surface of
the joint cavity. These are essentially the product
of a superficial infectious process. With the advance
of the inflammation, not only the articular cartilage but
also the adjacent bone structures break down and be-
come converted into fluid. The granulation tissue,
which extends also over the cartilaginous surface,
wears the latter away and destroys it. Then, when
the latter has been destroyed, the process extends to
the bone and establishes there the same destructive
changes as are observed in primary tuberculous os-
teitis. In the cavity of the joint, at this stage, there
is usually found a thinnish fluid, that looks like pus.
On examination, however, it is found to contain only
a few pus corpuscles, numerous products of disin-
tegration, detritus, and a few tubercle bacilli.

In favorable cases the granulation growths shrink

and become converted into connective tissue, and finally the joint heals, but with a permanent fibrous or bony ankylosis. As a result of the swelling of the soft parts in the neighborhood, the joint (knee or elbow) often presents the shape of a spindle. This appearance is also doubtless favored by the atrophy which takes place in the neighboring muscles, through lack of use.

In another group of cases the inflammation extends to the capsule of the joint, to the peri-articular connective tissue, and also to the skin covering these parts; and, when the latter is affected, it may show a great variety of secondary inflammatory alterations. Thus, for example, it becomes thin, smooth, and glossy (tumor albus; white swelling), and is often pierced by fistulous openings which afford escape to a thin abnormally colored pus.

Tuberculous joint disease is almost always a secondary disease of metastatic origin (due to hæmatogenous auto-infection). It develops from some older tuberculous disease located in the lungs, the lymph glands, or some other organ; it may also develop secondarily to a tuberculous synovitis or to a synovial form of tuberculous arthritis which was not preceded by tuberculous disease of the bone; or, finally—and this is more apt to be the case—it is a tertiary pathological phenomenon, due to the extension of a tuberculous osteitis (in circumscribed foci) from the neighboring bone to the joint (osteal variety of tuberculous osteitis). The primary and idiopathic occurrence of articular tuberculosis is an exceptional event.

As regards the relative frequency with which the different joints are affected, it can only be said that

the knee is oftener involved than any other joint; and
that next in the order of frequency come the hip and
elbow joints; these three joints being the ones af-
fected in eighty per cent of all the cases of tuberculous
joint disease.   The disease occurs more often in men
than in women, by reason of the fact that their occu-
pations subject them, in a greater degree, to all sorts
of injurious influences.   More than half of all the
cases (fifty-five per cent) occur in men who have
passed their twentieth year of life.

ARTHRITIS DEFORMANS; ARTHRITIS SICCA.

(Malum senile articulorum.   Plate 45.)

This insidious joint affection involves the synovial
membrane, and both the cartilaginous and the bony
structures; and it often attacks several joints at the
same time.   The most important pathological altera-
tions are proliferation of the synovial membrane;
fibrillation and wearing away of the cartilage; prolif-
eration of the latter to its margins, with some ten-
dency to ossification (as a result of which a new pro-
duction of bone, having the shape of a lip, surrounds
the margin of the joint); and, finally, the grinding
down of the surfaces of the joint, through the lack of
their protecting cartilages.   The inflamed synovial
membrane produces villous growths (synovitis villosa
or prolifera) or nodulated masses of tissue (arthro-
meningitis tuberosa).   The head of the femur under-
goes atrophy, and a sort of collar of proliferated
tissues forms around its margin, giving to it the
shape of a vegetable fungus.   In the larger joints a
serous exudation (hydrarthros) will often be found,

whereas in the smaller ones there may be no evidences whatever of any exudation (arthritis sicca). This form of articular inflammation occurs particularly in advanced life (malum senile), and oftener in women than in men; or it may develop as a result of some mechanical injury or of some disturbance of the nutrition (unfavorable hygienic environment, particularly in persons belonging to the poorer classes); or, finally, it may result from some unknown cause.

The articular affections which occur in connection with tabes dorsalis follow very much the same course as do those which are observed in arthritis deformans. Affections which are of a somewhat mild type and which involve several joints at the same time, are of frequent occurrence in tabes.

LOOSE OR MOVABLE BODIES IN A JOINT.

The osteal and osteochondral bodies which are found free in the cavity of a joint owe their origin either to arthritis deformans or to some mechanical injury which has caused the detachment of a portion of the articular surface. The *movable bodies of traumatic origin* consist of normal, living, articular cartilage, with a somewhat jagged surface (where the separation occurred) which is covered with connective tissue and cartilage. These portions which have become separated from the articular surface do not remain permanently free in the cavity of the joint, but soon form adhesions at some new point, and then undergo further changes. If a piece of bone has been torn off along with the cartilage, the broken surface of the former always secures for itself a new cover-

ing of cartilage and connective-tissue; the production of this covering taking its start from the point where the adhesions form. At some later date the connecting peduncle may first stretch and then finally give way altogether; and from this time forward all sorts of trouble may arise from this state of affairs in the joint. It is a curious fact that the cartilaginous portion of the piece which has been broken off from the articular surface continues to live, while the bony part dies.

Loose bodies of arthritic origin do not show the structure which is characteristic of normal articular cartilage.

The so-called *rice bodies* (corpora oryzoidea) originate from fibrinous material which has been precipitated from the synovial fluid. Thus, for example, in joints, sheaths of tendons, and bursæ mucosæ which are affected with tuberculous disease, all the rice bodies are essentially products of the coagulation of fibrin; the tuberculous disease generally playing a very mild part in the process. Some of the rice bodies originate from coagula which have formed in the synovial fluid itself, while others may be traced to products which have been deposited upon the walls of the synovial cavity, and which have, at least in some instances, become organized through the instrumentality of the cells of the part.

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